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Archives OF NEUROLOGY AND PSYCHIATRY

The Conduction of Pain Above the Level of the Thalamus Opticus

A. Biemond

Effect of Topical Hydrocortisone on the Electrocorticogram of the Cat

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Thomas S. Szasz

MARCH 1956

VOLUME 75 NUMBER 3

Pharmaceutical Treatment of Patients with Senile Brain Changes

Leopold Hofstatter, Abel Ossorio, Billie Mandl, Louis H. Kohler, Anthony K. Busch, and Ann Hyman

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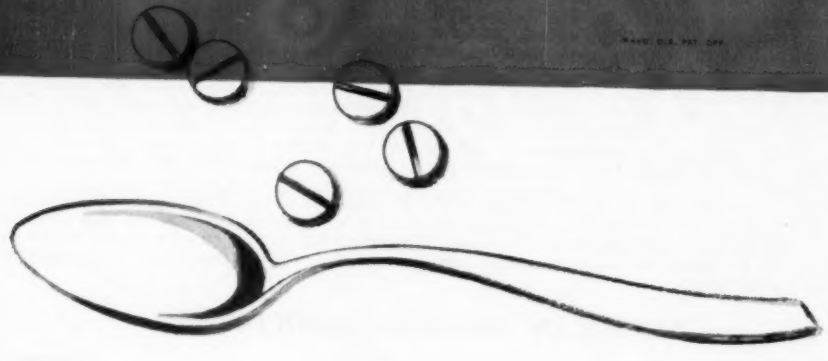

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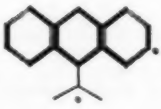

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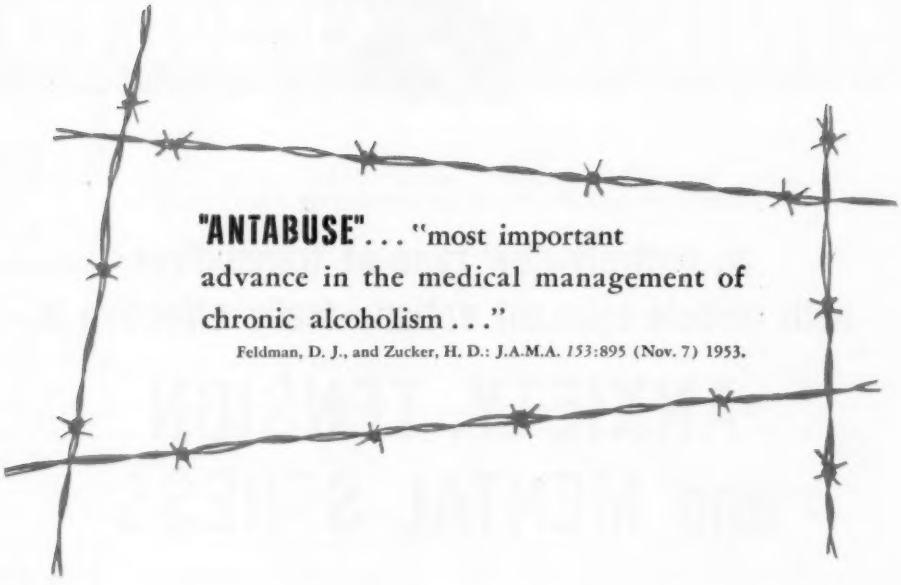
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The Conduction of Pain Above the Level of the Thalamus Opticus

PROF. DR. A. BIEMOND, Amsterdam

It is a clinically well-known fact that focal processes in the cerebrum above the level of the thalamus opticus may often lead to the occurrence of a crossed hemihypalgesia. The interpretation, however, of the disturbance in pain sense in such cases is frequently difficult, for repeatedly it is not so much—or not predominantly—the pain sensation itself that is disturbed; rather, indeed, it is the (secondary) experiencing of pain, by disturbances in attention or a change in psychic attitude toward pain in general. If this is the case, then a distinct localizing significance cannot be attributed to the symptom of hemihypalgesia. Besides, however, a relatively small group of suprathalamic focal processes showing crossed hemihypalgesia occur in which the pain conduction—or the primary identification of pain stimuli, as the case may be—seems to be essentially disturbed. This disturbance generally is transient in nature. For our problem, those cases especially are important in which only pain sense (and thermal sense), but not the gnostic sensibility, was disturbed. The number of cases appears to be remarkably small. I should like to mention only a few authors. Déjerine and Mouzon [1] (1914) described the case of a soldier who was hit by a bullet, causing an impression fracture in the left parietal area. Operative treatment was not established. On examination distinct hypalgesia and hypothermesthesia of the right half of the body appeared to exist, with the exception of the throat, neck, and face. Touch and proprioceptive sensibility remained completely intact. Foerster [2] (1936) indicates that in superficial lesions of the gyrus centralis posterior he repeatedly found crossed hypalgesia, but always temporary, and never as any permanent sensory loss. Kleist,[3] on the other hand, states that, in his experience of superficial cortical lesions, he had found 23 cases in which a localized parietal lesion appeared to have brought about mainly disturbances in pain and thermal senses. In eight of these cases the hypalgesia was the only sensory disturbance that could be established. Kleist, furthermore, thought it possible that parietal foci with disturbances in pain sense are localized more anteriorly than parietal lesions with disturbances predominantly in proprioceptive sensibility. He concludes from this that the projection of pain and temperature fibers on the sensory cortex presumably is different than that of the remaining sensory qualities. He is not able to be more precise, however.

The sensory disturbances caused by cortical bullet wounds during World War II were extensively studied, among others, by Marshall [4] (1951). This author reports 10 cases with superficial circumscribed cortical foci, resulting in distinct disturbances in pain

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From the Neurological University Clinic of the Wilhelmina-Gasthuis.

sensibility that were localized to subdivisions of an extremity or to a part of the face, and that could be clearly demonstrated even after years, showing an obviously organic character.

Marshall arrives at the conclusion that "the cerebral cortex is concerned with pain sensibility." At the same time, he again draws attention to the previously reported fact that disturbances in pain sensibility resulting from circumscribed cortical lesions evidently show less inclination to recovery than hypalgesia in consequence of extensive cortical lesions, in which apparently the crossed hemisphere is more capable of taking over the cortical projection.

After World War II the problem of pain conduction was brought up for extensive discussion at the International Congress on Neurology in Paris (1949), where the thalamus opticus and the surgery of pain were treated as main themes. The pain conduction from thalamus to cortex, however, was not then subjected to an exact scrutiny. Finally, in 1952, in an international symposium at Salzburg, the pain problem was again extensively discussed. The proceedings of this Congress contain a wealth of clinical, physiological, and neurosurgical data. The anatomical contributions describe precisely the pain-conducting fibers in peripheral nerves, spinal cord, brain stem, and thalamus. However, hardly anything about the thalamocortical projection is mentioned. Apparently, it is generally accepted that the pain-conducting fibers above the thalamus mix diffusely with the other sensory fibers and, like those, lead mainly to the gyrus centralis posterior. This cortical projection would be of less extent and would rank far below that of the proprioceptive sensibility in significance. Both the following clinicoanatomical observations caused me to doubt the above conception and to substitute another hypothesis.

REPORT OF CASES

CASE 1.—A few data relating to this case have already been reported at the International Congress on Neurology in 1949, in the discussion on the Thalamus Opticus. The complete description now follows. A 52-year-old ladies' hair dresser, previously completely healthy, on Dec. 10, 1945, suddenly had powerlessness of the left arm and left leg when dressing himself in the morning, though remaining fully conscious. Walking was possible. The general physician prescribed bed rest. After some four weeks he was mobilized. At that time the hemiparesis of the left side appeared markedly improved. As he began to walk more and more, he was troubled gradually by a pain in the left side and an unpleasant cold sensation of the left half of the abdomen. Subsequently, this pain extended by degrees over the entire left side of the body, where, at the same time, an increasing sensitivity to touch developed. During March and April, 1946, the patient was observed in a clinic at Enschede (Dr. Idema). Here, among other things, a lumbar puncture was performed, in which a normal constitution of the cerebrospinal fluid was found. Various medical and physical methods of treatment were tried, but these had little or no influence on the complaint. In the meantime, the strength of the left arm and leg had almost entirely returned. The patient was unable to start work. His former cheerful mood deteriorated to a feeling of depression. He repeatedly uttered suicidal intentions. Finally, on March 14, 1947, he was admitted to the Neurological Clinic at Amsterdam. From the written case report, the following data may be noted. Psychic disturbances were not noted. His affective reactions were little or not influenced by his continuous pain and hypersensitivity. He was always patient and cooperative, did not become excited, did not cry, was always friendly toward everybody, and behaved in other respects in a completely adequate manner. The examination of the internal organs likewise showed no abnormalities. As regards the neurological status, it may be mentioned first that a very slight paresis of the left arm and leg could be established. The arm reflexes were alike on the two sides; the reflex of Mayer could not be evoked on the left side, however. The abdominal reflexes on the left were somewhat lower than on the right; the patellar and Achilles tendon reflexes on the left were somewhat higher than on the right. The left plantar reflex was indifferent, whereas a normal flexion reflex could be elicited on the right. There were no pathological reflexes. The pupil and fundus showed no abnormalities. There was no hemianopsia. Smell and taste were undisturbed. The left half of the body constantly perspired slightly more than the right, but color and temperature were alike on the two sides. Otherwise the subjective and objective disturbances were entirely restricted to the sphere of sensibility. Subjectively, the patient complained constantly of a deep, "drilling" pain in the entire left half of the body, strongest in the left arm and the left half of the trunk. At the same time, a marked hypersensitivity of the entire left half of the body existed,

especially to gentle touch. When in his direct environment an abrupt movement occurred, the air current so caused gave an intense pain reaction. Gentle stroking with the fingers, even a slight shifting of the bed sheet, was extremely unpleasant to him. If, on the other hand, a strong pressure was exerted on the left, he was hardly troubled by it. The presence of warm food or drink in the cavity of the mouth caused a shivering of pain through the entire left half of the body. If, however, he consumed cold drinks or ice cream, this caused a passing sensation of pleasure. He was never able to smoke a cigar to the end, at any rate not when he kept it in his left hand, as he was wont to do. The curling of the smoke along his fingers caused intolerable pain. Objective examination of the sensibility revealed the following: The sense of touch was intact even when every tactile stimulus, especially when applied for a longer time, caused a very unpleasant sensation. The pain sense was greatly disturbed in the entire left half of the body, being most affected in the left arm and the left half of the trunk. Here he was unable to distinguish clearly between the head and the point of a pin. Now and then a pain stimulus caused a typical causalgia: After a certain latent period a painful burning sensation occurred, extending over the entire extremity. The thermal sense, likewise, was strongly disturbed on the left half of the body. Cold and heat on the left arm and the left half of the trunk were not distinguished. On the left half of the face and the left leg gross temperature differences could just be detected. As opposed to the marked disturbance of pain and temperature senses, the fact was established that the proprioceptive sensibility on the left half of the body was completely intact: Deep sensation, the sense of attitude, stereognosis, sense of vibration, sensation for figure writing, discrimination, and estimation of weights, all were undisturbed, with no demonstrable difference between left and right.

The clinical diagnosis was established as a special type of thalamic syndrome. It was attempted to decrease the subjective complaints by intravenous injections of high doses of sodium iodide. This treatment had no result; neither had physical therapy and psychotherapy. After ample consideration, it finally was decided to attempt to obtain improvement in the condition by excision of part of the right posterior parietal cortex. This operation appeared permissible in view of the results obtained in some cases of tenacious phantom pain. The operation was performed by Dr. Noordenbos on April 11, 1947. A small (2 cm. in diameter) piece of the cortex below the right interparietal sulcus, including the uppermost part of the supramarginal gyrus, was removed. It was interesting that prior to this operation it had been established that local injection of procaine into the posterior central gyrus appeared to have no influence at all on the hypersensitivity. After the cortical excision the hypersensitivity and painfulness of the left half of the body were diminished, but had certainly not disappeared. As a sequel to the operation, it appeared that a considerable disturbance in the proprioceptive sensibility of the left arm and hand existed, with complete astereognosis. After a few weeks this disturbance almost completely disappeared. Gradually the spontaneous pain and hypersensitivity of the left half of the body again increased. When the patient was dismissed from the clinic, on May 10, 1947, his condition, subjectively as well as objectively, was the same as on admission. On June 10, 1947, the patient committed suicide by means of gas.

Autopsy (Dr. Zeldenrust, Enschede).—Apart from the residua of a previous pleuritis, no abnormalities were found in the internal organs. On external inspection of the brain, it appeared that at the site of the operation, in the right parietal area, a small area existed where the dura mater was adherent to the surface of the brain. The brain was handed over to the anatomical laboratory of the Neurological Clinic for further examination. Here, in the usual way, a series of frontal transections was made. To our surprise, the thalamus macroscopically showed no abnormalities. It appeared, however, that a small focus of softening existed in the region of the right insula, parietal operculum, and adjacent part of the centrum semiovale. The operative lesion of the right parietal cortex appeared to extend into the underlying medullary substance. The middle part of the brain, in which the area of softening was situated, was serially sectioned bilaterally, the sections being alternately stained according to Weigert-Pal and Nissl methods. The frontal and occipital parts of the right hemisphere were prepared in the same way. The cerebellum and brain stem, which macroscopically showed no abnormalities, were not examined microscopically.

Histological Examination.—Study of the series from the rostral to the caudal pole revealed that the foremost tip of the focus was situated within the fissure of Sylvius on the basal plane of the ascending frontal gyrus, exactly where the frontal portion of the operculum passes into the operculum Rolandi. In this region occurred the anterior commissure and the anterior part of the globus pallidus. Slightly more posteriorly, the focus just extended into the underlying medulla, while the adjacent portion of the medulla (ascending frontal gyrus) showed a circumscribed area of demyelination. At this level, at the same time, a separate small focus of softening stood out in the cortex of the insula, extending into the claustrum but leaving the external capsule intact. At a more posteriorly situated level the opercular focus shifted from the cortex to the underlying medullary mass of the ascending

Fig. 1.—Focus of softening in the insula and parietal operculum. Weigert-Pal stain.

parietal gyrus, transecting this gyrus as a narrow, almost horizontal, cleft. The cortex at the convexity remained intact. The insular focus maintained its original position. At the level of the frontal pole of the anterior nucleus of the thalamus the insular focus appeared to have expanded upward, thus coming into continuous relation with the opercular focus (Fig. 1). The most laterally situated fibers show demyelination. At the level of the development of the anterior part of the lateral nucleus of the thalamus, the bridge between the two foci was interrupted. Here were visible the insular focus, extending into the innermost part of the opercular cortex, and, separately, a few small cavities of the medulla of the ascending parietal gyrus. At a level through the middle of the thalamus only the insular focus was still visible. Local demyelination in the medullary mass directly over the insular focus remained, however. At the level of the oral pole of the external geniculate body a narrow, dense bundle of demyelinated fibers (Fig. 2) began to stand out in the vertical part of the internal capsule (pars posterior). This bundle of fibers obviously originated from the demyelinated portion of the medullary mantle of the operculum. In posterior sections it could clearly be established that the bundle was directed toward the posterior part of the latero-ventral nucleus of the thalamus. Furthermore, it appeared that at the level of the greatest extent of the external geniculate body yet another small focus of softening appeared basally in the opercular medulla. At the level of the caudal part of the external geniculate body this opercular focus apparently had shifted toward the cortex, whereas the insular focus at this level extended slightly into the internal capsule, just anterior to the point where this became transient into the internal capsule. In the more posteriorly made sections, in which the pulvinar became visible, both foci appeared to have vanished. Much more posteriorly, at the level of the splenium of the corpus callosum, two foci were again encountered, viz., (1) a super-

Fig. 2.—The same focus as that in Figure 1, at the level of the oral pole of the external geniculate body. A narrow, dense bundle of demyelinated fibers begins to stand out in the vertical part of the internal capsule (pars posterior). Weigert-Pal stain.

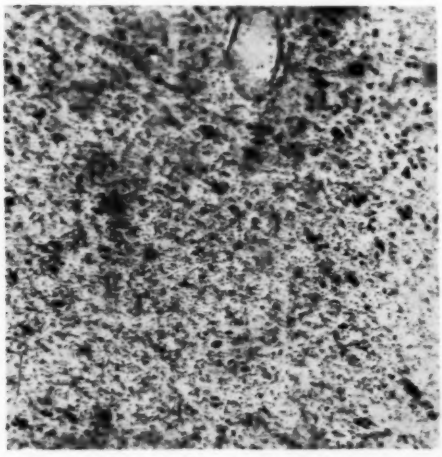


Fig. 3.—Loss of cells in the posterolateral ventral nucleus of the thalamus. Nissl stain.

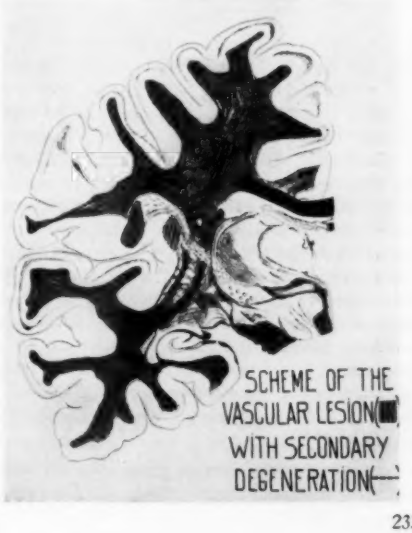
ficial and local destruction of the inferior parietal gyrus (without visible secondary degeneration), caused by the operative procedure of March, 1947, and (2) an irregularly shaped, hemorrhagic, deep focus of softening in the medulla of the second temporal gyrus, reaching into the dorsal segment of the optic radiation. The impression was given that the latter focus was a recent area of softening, arising after observation in the Neurological Clinic.

By means of the Nissl serial sections, we are, at the same time, able to orientate ourselves exactly about the cytological relationships. In the cortex, the parietal operculum especially appeared to show a considerable, at some places maximal, loss in cells, even beyond the area of softening proper. The posterior central gyrus was intact except in the very lowest part, where a moderate cellular loss was observed in the deep cortical layers. The changes in the insular cortex were restricted to the softened area. In the thalamic nuclei, these all showed a normal structure, with the exception of the posterolateral and posteromedial ventral nuclei, where a marked cellular loss was observed (Fig. 3). The impression was created that this loss had occurred more or less in cell groups, between which groups of normal cells had remained intact. There was no marked difference between the lateral and the medial portion of the posterior ventral nucleus. However, the cellular loss did appear distinctly more marked in the caudal portion than in the proximal portion of the above-mentioned nuclei. In the pulvinar, subthalamic body, centrum medianum, and the striate body no cellular abnormalities were found.

Summary.—A 52-year-old man, following a slight vascular accident, developed a typical hyperpathia and, at the same time, spontaneous painfulness of the entire left half of the body. On examination it appeared that pain and temperature senses were markedly disturbed on the left, whereas the proprioceptive sensibility had remained entirely intact. A cortical excision at the site of the right inferior parietal lobe had no effect, and left no lasting functional loss. Death occurred later, from suicide, about one-half year after the onset of the disorder. Autopsy revealed the following main abnormalities (Fig. 4): (1) three confluent small foci of softening in the opposite sides of the insula and parietal operculum; (2) an area of secondary degeneration in the ventral part of the thalamocortical radiation, traceable via the internal capsule into the lateroventral nucleus of the thalamus, and (3) a marked cellular loss, most pronounced caudally in the posterolateral and posteromedial ventral nuclei of the thalamus. Unimportant accessory findings were an operative lesion on the right inferior parietal lobe and a recent softening in the medulla of the right second temporal gyrus.

Case 2.—A 45-year-old warehouse man was admitted to the Neurological Clinic on Feb. 13, 1950. From the history it appeared that during the past few months he had been troubled by slight headache and vertigo when stooping down. Three weeks prior

Fig. 4.— Scheme of the lesion in Case 1.



to admission the headache became worse, being localized over the right eye. Starting from that region, the pain radiated to the right side of the neck. A few days later the patient also began to vomit and was confined to bed. Paralytic symptoms did not occur, but the patient became gradually somewhat dull. On examination bilateral papilledema was found; in addition, there was a distinct bradyphrenia with slight disorientation for time. A minimal paresis of the left facial nerve and the left arm existed, but the reflex relations were normal. It was striking that pain and temperature stimuli were not clearly observed on the left half of the body. The proprioceptive sensibility that could then be examined adequately appeared to be intact. The psychic reaction to pain stimuli in general appeared undisturbed. The clinical diagnosis was established as tumor of the brain, presumably localized to the right parietal area. On ventriculographic examination, a considerable displacement of the lateral ventricles to the left was observed, the left ventricle appearing much wider than the right one. The displacement to the left was even more pronounced in the third ventricle, however. On operation, on Feb. 15, 1950 (Dr. de Grood), a large right frontoparietal flap was made. At that time only signs of an increased intracerebral pressure were observed, but no tumor was seen. On deep puncture, likewise, no tumor was sounded. A decompression was performed. After the operation the condition of the patient grew worse. He died on Feb. 22, 1950, with a high fever and symptoms of pneumonia.

Autopsy revealed bilateral pneumonia, mucopurulent bronchitis, and bilateral fibrous adhesive pleuritis. The brain was distinctly flattened, with signs of a pressure cone at the tonsils of the cerebellum. On transection of the brain a small tumor (3 cm. in diameter) was found in the region of the right external capsule, the tumor being round and sharply defined from the adjacent areas. The brain was handed over to the anatomical laboratory of the Neurological Clinic for further examination. The middle part of the brain, containing the tumor, was serially sectioned and stained according to the Weigert-Pal and Van Gieson methods. Histological examination revealed the following: In the interior of the right cerebral hemisphere a globular, sharply defined tumor, 2.5 to 3 cm. across, was found. The entire center of the tumor was necrotic. Only along the edge was readily recognizable tumor tissue found, sharply delineated in regard to the surrounding brain tissue, but not showing a proper capsule. The tumor undoubtedly was a glioma, not very polymorphic in nature, and with relatively abundant fiber formation. Further specification of the glioma is impossible because of the absence of a specific stain. The tumor was bounded laterally by the insula, dorsally and dorsomedially by the striate body, medioventrally by the cornu ammonis formation, and ventrally and ventrolaterally by the white substance of the temporal lobe (Fig. 5). The insula clearly had become displaced toward the outside, and the striatum dorsomedially. The internal capsule was also medially displaced and, at the same time stretched out in a longitudinal direction. The greater part of the right lateral ventricle had been compressed; the left ventricle was widened. The right striate body with its subdivisions was well recognizable, but there existed partial demyelina-

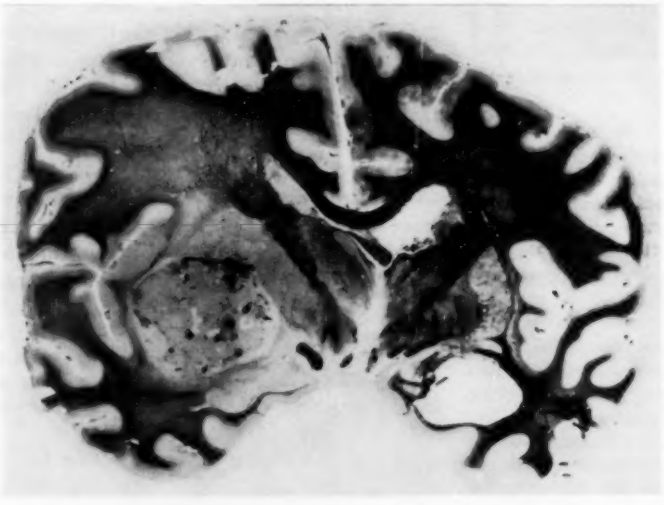


Fig. 5.—Position of the tumor in Case 2. Weigert-Pal stain.

tion of the putamen, as well as of the pallidum. The right internal capsule was thinner than the left one, probably through stretching, but without marked loss of color. The external capsule could not be recognized well; neither could the claustrum. It seems probable that the tumor originated from the claustrum and very gradually had led to a displacement of the adjacent parts of the brain. In the posterior part of the series of sections the tumor was situated almost directly against the internal capsule, which, however, here likewise showed no loss of color. It now appeared clearly that the radiating fibers of the ventral part of the right corona radiata were for the greater part demyelinated. This was particularly marked in the neighborhood of the parietal operculum (Fig. 6). The cortex of the insula and operculum showed no distinct abnormalities, whereas the thalamus in its various subdivisions also appeared normal.

Fig. 6.—Transverse section on a more posterior plane in Case 2. There is demyelination of the ventral part of the corona radiata, particularly marked in the neighborhood of the parietal operculum. Weigert-Pal stain.

Summary.—A 45-year-old man a few months prior to admission developed symptoms of brain tumor. As the sole distinct local symptom, hypalgesia and hypothermesthesia of the left half of the body were observed. Operative exploration of the right parietal cortex gave a negative result. At autopsy a circumscribed tumor was found between the insula and the internal capsule. The tumor had nowhere led to infiltration of the surrounding structures, but had produced only displacement. On serial section, it appeared that the fiber radiation in the ventral part of the corona radiata had for the greater part disappeared at the level of the parietal operculum.

CASE 3.—The third case so far is of less significance, in that, besides complete analgesia of the upper part of one-half of the body a considerable disturbance of the deep sensibility existed. The peculiar localization of the disturbance in pain sense, however, and the extent of the cortical focal process, render this case worth reporting.

A 78-year-old man, previously healthy and sound of mind, on Jan. 22, 1955, called on an acquaintance in order to have a game of cards. On arrival, he had to be helped with taking off his coat, since the left arm suddenly appeared to have become strengthless. Shortly afterward, when he was about to seat himself, his left leg suddenly gave way, so that he dropped to the floor. Consciousness remained completely undisturbed. Subsequently he was brought to his house by means of a motor ambulance, and two days later he was admitted to the Neurological Clinic. On admission he gave a somewhat dull, sleepy impression, but otherwise he was compos mentis. The temperature was normal; the pulse was well filled; there was no tachycardia, but some irregularity; the blood pressure was 150/90. There were no symptoms of cardiac decompensation; the heart, however, was distinctly enlarged, a systolic murmur being audible over all ostia. 1. Neurologically, the patient showed a left hemiplegia (including the facial nerve), in the course of observation gradually passing clearly into hemiparesis; especially in the left leg partial recovery clearly occurred, a paresis remaining, however; in the left arm, only a slight motility in the shoulder and elbow joints returned; the hemiparesis was clearly hypotonic; the tendon reflexes were stronger on the left; the abdominal

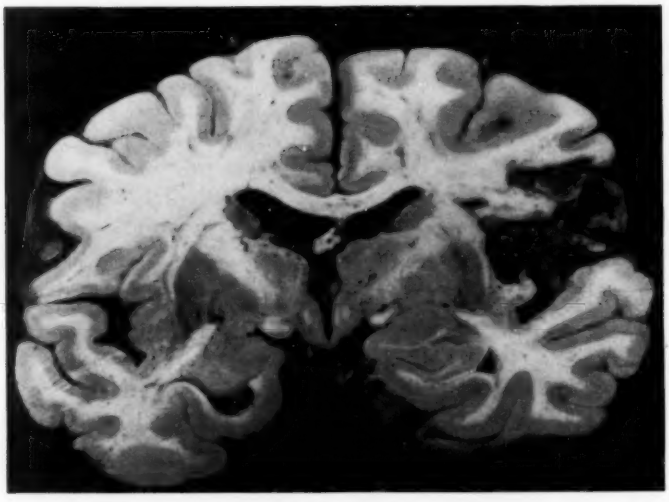


Fig. 7 (Case 3).—Circumscribed focus of softening in the lower parietal area and the operculum Rolandi.

reflexes were absent on the left, whereas in the left foot a distinct Babinski reflex could be evoked. 2. Whereas it initially appeared that a left hemianopsia existed, it soon was evident on more accurate examination that in this case a disturbance in attention was principally involved; toward the end of the observation period one could hardly speak of hemianopsia, only a slight restriction in the left upper quadrant remaining. 3. Extremely important, as well as constantly apparent, were the disturbances in sensibility, pronounced for all qualities in the left half of the body. As the most important finding, it should be mentioned that the pain sense in the left half of the face and head, in the left half of the trunk, and in the left arm was entirely absent and remained so during the period of clinical observation (one month), during which period the patient was repeatedly examined. In the left leg hypalgesia existed, whereas in the left foot definite hyperalgesia was found. The temperature sense in the left body-half was analogously disturbed, except that in the left foot a hypersensitivity to thermal stimuli was lacking. The senses of touch, posture, and discrimination, as well as deep sensitivity, were likewise considerably disturbed on the left, but were not completely absent. For these qualities no difference between the upper and the lower part of the body was established, as was the case for the

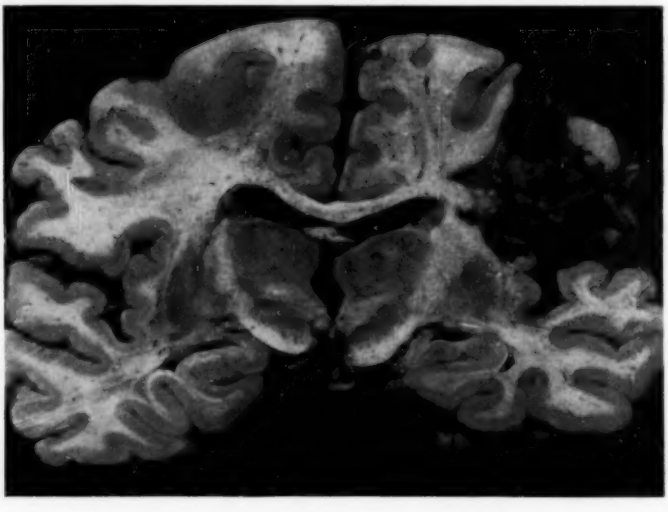


Fig. 8 (Case 3).—The same focus at a more posterior level.

pain sense. The left corneal reflex, although decreased, could still be distinctly elicited. The stereognostic function could not be examined because of the paralysis of the left hand. Anosognosia was not present. The patient appeared to be conscious of his paralysis. On Feb. 25, that is, exactly one month after admission, when the patient had just been mobilized and had been sitting in a chair for an hour, he suddenly became very cyanotic and died within a few seconds.

At autopsy it appeared that death had resulted from a pulmonary embolus. In addition, there was myodegeneration of the heart and considerable sclerosis of the aorta.

The brain, on external inspection, showed a small focus of softening in the lower part of the right central gyrus, extending into the Sylvian fissure. In frontal sections of the brain a circumscribed focus of softening was found, obviously caused by a thrombotic (or embolic) occlusion of the anterior parietal branch of the right middle cerebral artery. The area of softening was so well defined in the fixed brain that a macroscopic description of the unstained preparation was sufficient. When the extent of the focus was followed in a frontal-caudal direction, the lesion appeared to commence in the cortex of the insula at the level of the maximal development of the anterior commissure. More posteriorly, at the level of the commencement of the thalamus, the focus passed into the cortex of the operculum Rolandi and into the lower parietal gyrus (Fig. 7). More posteriorly, at the level of the maximal development of the thalamus, the focus reached its maximal extent, now comprising the upper part of the insula, the operculum Rolandi, and the lower parietal gyri, including the adjacent portion of the centrum semiovale, up to the intraparietal sulcus (Fig. 8). Even more posteriorly, the focus rapidly decreased in extent. At the level of the pulvinar only a small softening could be seen subcortically in the lower parietal gyrus.

Summary.—A 78-year-old man, subsequent to a vascular insult, developed acute left-sided hemiplegia, which after a few weeks showed a partial recovery, and left-sided hemianesthesia, remaining constant in the course of the month of observation. The gnostic sensibility was considerably disturbed over the entire left body-half but was not completely absent. The pain and temperature senses were completely absent in the left half of the face and head, the left arm, and the left half of the trunk, and only partially disturbed in the left leg, whereas in the left foot a clear hyperalgesia existed. Autopsy revealed a circumscribed focus of softening in the lower parietal area and the operculum Rolandi. It is assumed that this cortical and subcortical focus had caused a complete loss of the pain perception of the crossed body-half (face, arm, and trunk).

COMMENT

The first case presented might, as regards the subjective symptoms, be taken clinically as a typical example of the thalamus syndrome. The constantly present, deeply localized, drilling pain in the entire left half of the body, and the pronounced hyperpathia (for gentle touch) were extraordinarily impressive. These symptoms, after failure of the treatment, finally led to suicide. As regards the objective symptoms, the picture was not typical, however. The disturbance in sensibility was exactly the reverse of that usually encountered in the classical thalamus syndrome, for the sensibility for pain and temperature was considerably disturbed on the left, whereas the protopathic sensibility had remained completely intact. If, however, the spontaneous unilateral pain is taken to be the most essential element of the thalamus syndrome, the case is another instance of this syndrome caused by a focus outside the thalamus. That a parietal focus might cause this syndrome was shown years ago by Lhermitte and Ajuriaguerra [5] (1935) and by Davison and Schick [6] (1935). That a pontine focus may also produce the same syndrome was shown, among others, by Lhermitte, Lévy, and Trelles [7] (1935). In lesions at a lower level in the brain stem, and even in spinal lesions, obviously through pressure on the spinothalamic tract, typical unilateral pain may occur, as appears from the paper by Alajouanine and Thurel [8] (1949). Finally, that this syndrome may develop from a process in the thalamocortical tract had been reported in the literature, but so far anatomical verification had been insufficient. Our case may be presented as a typical example.

The peculiar objective disturbances in sensibility in our case enable us to remark on the possible etiology of unilateral pain. Many authors—e. g., Foerster [9]—assume that the proprioceptive sensory system at the various

levels of the central nervous system would exert an inhibiting action on the pain-conducting system. Loss of this inhibition would be one of the main causes for the development of a hyperpathia in general. Since in our case the proprioceptive sensibility in the painful half of the body had remained completely intact, such an explanation certainly does not hold. A loss of inhibiting corticothalamic fibers, as indicated by Head and Holmes[10] as an explanation of thalamic pain, cannot be applied in our case. Even apart from the fact that such fibers probably do not terminate in the posterior ventral nucleus, the focal process in our case was much too circumscribed to render such a functional loss plausible. I shall refrain from discussing the other theories, launched in the course of the years, on the origin of thalamic pain. They likewise suffer from the fault of being too complicated. I think, therefore, that our case, through the peculiar localization of the focal process, is a clear confirmation of the opinion of Le Gros Clark,[11] expressed in 1949, and quoted here:

> In the past the discussion on these paraesthetic phenomena has been mainly concerned with suggestions that they are either due to the irritative effect of local lesions or to the interruption of cortico-thalamic inhibitory fibres. It now appears more probable that they may be simply the result of a disturbance of the normal pattern of thalamo-cortical connections, and thus parallel the abnormal sensory phenomena which we now know to be produced by a disturbance of peripheral pattern of cutaneous innervation.

Besides its casuistic significance, however, our case seems to have value for the approach to the problem of cortical pain projection. It seems permissible to correlate the observed hemihypalgesia with the cellular loss in the posterior ventral nucleus, the retrograde degeneration in a circumscribed fiber system of the posterior portion of the internal capsule, and the focal process in the medulla and cortex of the parietal operculum. As regards the last, the findings are corroborated by those in the second case. In this case a circumscribed tumor existed, arising from the external capsule and having produced a curving out of the internal capsule and local demyelination in the parietal operculum. Clinically, in this case a distinct hemihypalgesia existed. All this, therefore, would lead to the conclusion that above the level of the thalamus a closed, circumscribed pain (and temperature)-conducting fiber system exists, with a cortical projection of its own.

Such a conception, at first view, seems to meet with great practical objections—clinical, physiological and anatomical. I shall enumerate the principal objections and try to disprove them. First may be pointed out the relative rarity of unilateral disturbances in pain in suprathalamic processes, and the fact that mostly these are transient in nature. However, this rarity is probably only apparent, since in many cases a fusion with psychogenic factors makes an adequate analysis of the sensory disturbances difficult or impossible. On the other hand, the pathway suggested by us is situated in a region that is relatively rarely attacked by vascular processes, and that appears to be sufficiently protected against the effects of skull trauma. The transient nature of the disturbance can be readily explained by the fact that each spinothalamic tract becomes connected with both posterior ventral nuclei of the thalamus, although the homolateral conduction appears to be dominant. A functional take-over by the other cerebral hemisphere, therefore, can be explained anatomically, without further discussion. This may come into effect very rapidly, as, indeed, experiences with hemispherectomy teach us, which are now being applied more and more. That such a take-over may also require a long time is shown by our case history.

Physiologically, the objections appear even stronger. It is known that in the experimental animal peripherally applied pain stimuli cannot be recorded from the cortex, whereas tactile stimuli appear to show a very distinct local projection on the cerebral cortex. In man, likewise, it has been possible to establish that pain stimuli either do not cause any reaction at all in the electroencephalogram, or, when the strength of the stimulus is increased, produce a diffuse slowing down of the rhythm in all leads (*réaction d'arrêt*).

Moreover, electrical stimulation of various cortical areas in man, according to the extensive experience of Foerster,[2] nowhere evokes pain sensations. Finally, it seems unacceptable that such a vitál function as the sensation of pain would be "localized" in such a circumscribed region of the cortex. Nevertheless, we become fully conscious of pain. In the first place we distinguish between the head and the point of a pin not by our power of discrimination, but above all, by the very special feeling of pain produced by the point of the pin. We are able to define exactly the type of pain, and also to keep it exactly in our memory. These facts seem compatible only with a cortical projection. That a pain stimulus cannot be recorded electroencephalographically may be due to various factors, concerning which at the moment one can only speculate. First, it may be possible that the supposed local cortical reaction is completely covered by a diffuse reaction, and thus escapes electroencephalographic analysis. In the noteworthy treatise by Hoff, Paterski, and Wanko [12] (1953), it is expounded how in pain conduction two principal physiological mechanisms of the brain come into action, blended into one harmonious whole in the healthy man. The one mechanism is the proper sensation of pain (ἄλγος), conducted by the posterior ventral nucleus to the sensory cortex. The other mechanism is the experience of pain (πάθος), effected by the stimulus being conducted simultaneously through the diffuse thalamic system of nuclei, and from there projected on the entire cortex. This pain experience is further stressed by the fact that each thalamocortical conduction is followed by a corticothalamic stimulation and vice versa. In establishing the final pain sensation, therefore, sensory cortex, total cortex, and thalamus form an indissoluble unit. The difficulties of electroencephalographic recording may simply be a result of the specific nature of the stimulus. By a refined technique, especially by frequency analysis, these difficulties may perhaps be overcome.

Finally, it is of importance that one look for the local response in the cortical area whither the pain stimulus is conducted in the first instance. I assume this to be not the posterior central gyrus but the parietal operculum.

Finally, as to the anatomical difficulties. Glees,[13] in his recent study (1953), reports his investigations concerning the projection of the spinothalamic tract on the posterior ventral nucleus of the thalamus in the monkey. At the same time, he performed a fiber analysis, in which he was struck by the small number of myelinated fibers (1500-2000) encountered in the mesencephalic part of the spinothalamic tract. One wonders whether this tract, appearing to have given off the majority of its fibers at the upper part of its course, really fulfils the role of transferring pure pain sensation to another closed neurone. This objection cannot be invalidated without further discussion. In the meantime it is possible, as was also suggested by Glees, that the spinothalamic tract also contains unmyelinated fibers. It is of further importance that this tract, on termination in the posterior ventral nucleus, shows a projection as regards body subdivisions corresponding to the medial lemniscus. There exists the possibility of the nerve-termination subdivision dividing in the thalamic nuclei, so that a single fiber transfers the stimulus to several cells. Further refined histological investigation will have to determine this point. In any case, it is not a priori impossible that a very narrow pain-conducting system transfers the specific stimulus to a subsequent neurone, which seems to have a considerably larger extent.

However, there are also positive indications, apart from the data we have supplied, that support the hypothesis of circumscribed pain conduction and projection on the parietal operculum. Let us state at the outset that the course of the thalamocortical sensory projection fibers is not yet precisely known. In the monkey, Sachs [14] found that these fibers are directed toward the posterior central gyrus and, to a slighter degree, toward the motor cortex. Minkowski,[15] on the other hand, found that the fibers of the posterior part of the posterior ventral nucleus are di-

rected toward the supramarginal gyrus, partly also toward the parietal operculum. It may be indicated briefly here that Brodmann has pointed out that the posterior central gyrus extends into the Sylvian fissure and forms a part of the operculum Rolandi. Even more important are the relationships in the case "Longery," as described by Déjerine.[16] This was a case of hydrocephalus in a child who died at the age of 2½ months and in whom the entire cerebral convexity appeared absent. Déjerine, in this case, was able to observe accurately the course of the thalamocortical fibers, at least

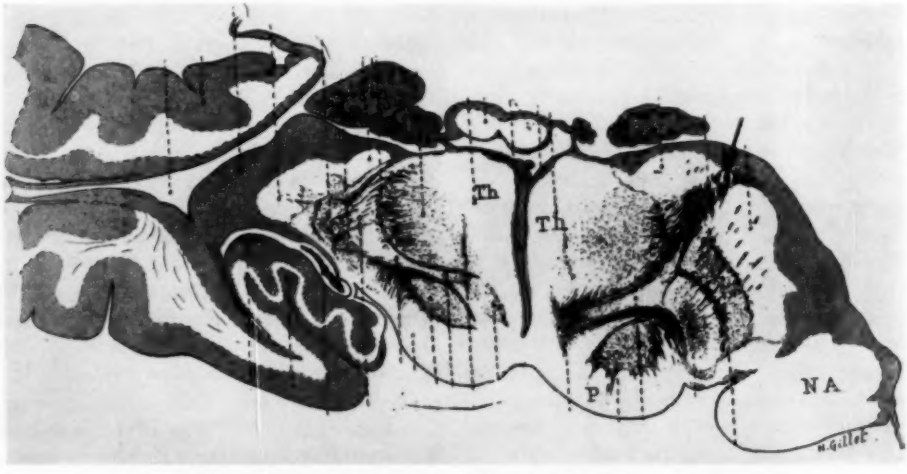


Fig. 9.—Course of the thalamocortical fibers in Déjerine's case "Longery."

in the first part of their pathway. These fibers first made their way between thalamus and globus pallidus, then between caudate nucleus and putamen (Fig. 9). The course of these fibers appears to correspond exactly with that part of the thalamic stalk that appeared degenerated in our Case 1. It is likely that this group of fibers in Déjerine's case was developed early and must be taken as the pathway system for "paleosensibility."

The remarkable syndrome of pain asymbolia, likewise, is of importance in our problem. The number of observations dealing with this is restricted, and the pathophysiological explanation is still obscure. Nevertheless, it is a characteristic picture, which must be striking to the clinician. It has the following typical features: 1. The patient remains indifferent to any pain stimulus and does not show a trace of antipathy to the examiner. 2. The defense reaction is slight or completely absent. 3. There is also no reaction to threatening gestures, at any rate not adequate. The syndrome as a rule develops suddenly, after a vascular accident. Following their original paper, Schilder and Stengel[17] (1928) later made several reports (1930, 1931), while further cases have been described by Pötzl and Stengel[18] (1936), Rubins and Friedman[19] (1948), and Hecaen and Ajuriaguerra[20] (1950). In most cases, with the pain asymbolia, a sensory aphasia existed simultaneously, with apractic symptoms and disturbances in the body scheme. Only in two cases was a comprehensive anatomical verification by means of serial sections made (Schilder and Stengel, 1930, and Pötzl and Stengel, 1936). In both cases multiple areas of softening were found in the left cerebral hemisphere (partly also in the right), in the supramarginal gyrus, in the temporal lobe cortex, and in the insula, with the parietal operculum. The authors assume that the first-mentioned focus must be of special significance for the development of the syndrome, for which opin-

ion, however, no cogent evidence is given. In any case, this syndrome is a complex one which must be approached not only neurologically but also psychologically. It seems certain, however, that a primary disturbance in pain sensation forms part of this syndrome, the more so since in the majority of cases the pain asymbolia appeared to be most pronounced in one-half of the body. Neither the temporal focus nor the focus in the supramarginal gyrus can be held responsible for this.

It is attractive to draw the so-called secondary sensory area into this discussion. This was first discovered in various experimental

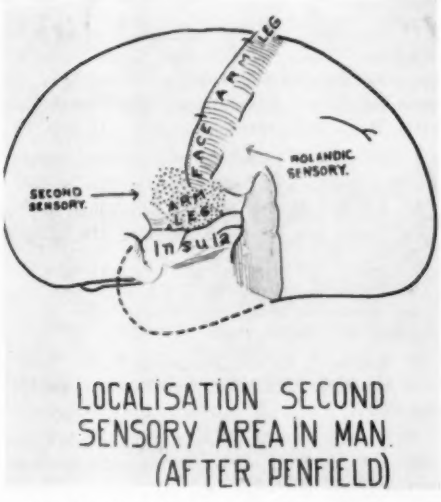


Fig. 10.—Localization of second area in man (reproduced from Penfield, W., and Rasmussen, A. T.: The Cerebral Cortex of Man, New York, The Macmillan Company, 1950).

animals (Adrian, Woolsey). Apart from the known primary area, a secondary area was found, appearing to receive stimuli from both halves of the body, but mostly from the contralateral part. According to the investigation of Woolsey,[21] in the monkey the secondary area appears to be situated ventral to the primary area, and for an important part within the Sylvian fissure. In man this zone also appears to exist. Penfield and Rasmussen,[22] in 1950, reported 10 cases in which this area could be defined by electrical stimulation of the cortex. In 1954 the number of observations had increased to 16. The secondary area is situated lower than the primary, and more anteriorly it is found partly within the Sylvian fissure, consequently including part of the parietal operculum (Fig. 10). In this area a projection of the body subdivisions also exists: The face is represented at the top; the arm below that; the leg lowest. On stimulation in the relative body part, numb sensations occurred, as well as prickings, and sometimes also a sensation of movement or of powerlessness. Furthermore, it was interesting that stimulation of this area caused a suppression of the entire cortical activity. It seems likely that extirpation of the secondary area does not cause symptoms of functional loss. As to the function, Penfield can only speculate. Perhaps it has something to do with movement.

Now that this area, within the fissure of Sylvius, appears almost to correspond with the parietal operculum, our three observations become even more significant. It will have to be demonstrated further physiologically that actually a cortical pain projection is established in this well-concealed and protected region.

SUMMARY

The case is described of a 52-year-old man who, after a vascular accident, showed "thalamic" pain and hyperpathia of the left half of the body, this being so intense that it finally led to suicide. It was noteworthy that pain and thermal sensations were distinctly disturbed on the left, whereas the proprioceptive sensibility was completely intact. At autopsy a complex of small confluent foci of softening was found in the cortex and the medulla of the right parietal operculum and in the cortex of the insula. A retrograde degenerated fiber bundle could be traced via the posterior part of the internal capsule into the posteroventral nucleus of the thalamus, where a marked cellular loss had occurred. A distinct hypalgesia likewise existed in the second case, that of a circumscribed tumor, which had originated in the external capsule and led to demyelination of the medullary radiation to the parietal operculum. In the third case, that of a

superficial focus of softening in the lower parietal gyri and the parietal operculum, a very marked disturbance in pain sensibility was found established in the crossed body-half, excluding the leg. In the discussion, attention is drawn to an observation by Déjerine and to the anatomical findings in the syndrome of pain asymbolia. Finally, it is pointed out that the so-called secondary sensory area, defined in man by Penfield, probably corresponds to the area which in the first clinicoanatomical observations may be considered as lost. It is accepted as a working hypothesis that at this site, and not in the posterior central gyrus, conscious pain sensation is established.

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Effect of Topical Hydrocortisone on the Electrocorticogram of the Cat

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Studies of the effect of corticotropin (ACTH) and adrenocortical hormones on the electrical activity of the brain are numerous. The effects of corticotropin, cortisone, and hydrocortisone are similar, and they are the reverse of those of desoxycorticosterone.*

Cortisone decreases brain excitability in normal rats, as measured by electroshock seizure threshold and pentylenetetrazol (Metrazol) seizure threshold.† It counteracts the depressive action on brain excitability of desoxycorticosterone [2] in normal and adrenalectomized rats. In patients receiving cortisone and in subjects with normal EEG traces, it causes an increase of alpha frequency.[4] In adrenalectomized rats and patients with an abnormal EEG or Addison's disease, the slow EEG returns to normal under cortisone treatment.‡

Hydrocortisone administered intravenously increases theta activity in normal and epileptic subjects, with increase in bilateral spike-wave discharges.[7] The same abnormality has been observed in patients under cortisone treatment.[8]

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* References 1 and 2.

† References 1 and 3.

‡ References 5 and 6.

The action of the hormones on brain excitability has been explained (*a*) as a direct action of the hormone on the brain electrical activty,§ and (*b*) as an alteration of cerebral blood flow and metabolism. Adrenocortical hormones have no effect on brain enzymes in in vitro experiments.[10] Patients treated with cortisone have an increased cerebral vascular resistance, but the mean cerebral blood flow remains unchanged, as does oxygen utilization.[11] In adrenalectomized animals cortisone restores the blood flow to normal and, as a consequence, increases oxygen utilization,[12] causing the slow EEG to return to normal. The same mechanism may be responsible for the restoration of the EEG in the patients treated for Addison's disease.

Changes in the electrical activity of the brain have also been explained on the basis of alteration of the electrolyte metabolism of sodium and potassium. Torda and Wolff [13] reported retention of sodium in extracellular spaces in prolonged corticotropin treatment. Lack of changes in the intercellular sodium in spite of alterations of plasma sodium level have also been reported.‖ In intact and adrenalectomized rats the electroshock seizure threshold is parellel to the concentration of extracellular sodium.¶ In adrenalectomized rats changes in the plasma sodium level are directly correlated with changes in the electroshock seizure threshold.[14]

Leiderman and Katzman [15] attributed the changes in the EEG to the restoration of the potassium equilibrium by cortisone. Intracisternal hydrocortisone has been reported to

§ References 8 and 9.

‖ References 5 and 12.

¶ References 1 and 9.

produce no effect on the EEG in anesthetized cats.[16]

The effects of hydrocortisone on inflammation in the subarachnoid space,[17] its clinical use as a co-adjutant intrathecally in the treatment of tuberculous meningitis,# and the topical application of single doses of hydrocortisone acetate to wounds of the brain in cats, with depression of the inflammatory reaction and diminished cerebral cicatrix,[20] suggested that the hormone could be used clinically in traumatic and inflammatory conditions of the brain and meninges.

A study was made of electrocorticogram when hydrocortisone was applied directly to the cortex. The effect of nicotinamide, which increases the solubility of hydrocortisone 10 times in a freshly prepared solution,* was also studied.

METHOD

Cats of an average weight of 2.5 kg. were used for this experiment. Tracheotomy was performed with the animal under light anesthesia. Small doses of curare were given intravenously, and the animal was placed on artificial respiration. The cerebral cortex was exposed, and the electrocorticogram (ECG) was obtained. The recordings were made with a six-channel Offner ink-writing electroencephalograph, using two silver-wire monopolar electrodes placed in the somatosensory (SS) and the auditory (A) areas.

Hydrocortisone acetate was applied to the cortex in powder form,† in solution (50 mg. in 500 cc. of Elliott's Solution A,[21] ‡) and in combination with nicotinamide (25 mg. in 25 cc. of 5% nicotinamide in Elliott's Solution A).

Nicotinamide was applied to the cortex in powder form, alone and in combination with cortisone powder, and in solution (5% in Elliott's Solution A).

One animal was operated upon, and 30 mg. of hydrocortisone acetate powder was applied to the cortex. The craniectomy was reopened four days later for recording.

Records of the different effects of the powders and of the solutions were taken several times in the same animal. A total of 10 animals were used. Photographs of the vessels were taken and the changes correlated with the ECG. Specimens of the cortex to which the hormone and drug had been applied were taken for pathological studies.

RESULTS

I. Alterations in the ECG

Topical application of saline and of Elliott's Solution A at body temperature caused a brief excitatory effect, decrease in amplitude, and increase in background frequency. These effects did not last longer than 30 seconds, depending on the state of arousal of the animal when the drug was applied, the temperature, and the pH control (Fig. 1).

Effects of Hydrocortisone and Nicotinamide.—(*a*) Effect on Resting Rhythms: After a base line had been obtained, hydrocortisone acetate was applied topically in powder form. A continuous tracing was run for more than one hour without marked change of electrical activity in frequency or amplitude (Fig. 2). When the hydrocorti-

References 18 and 19.

* Laurie, J. H.: Personal communication to the authors.

† Hydrocortisone acetate and 5% nicotinamide in Elliott's Solution A were supplied by Merck & Co., Ltd., Montreal.

‡ Elliott's Solution A has the following formula: NaCl, 86 gm.; KCl, 3 gm., $CaCl_2$, 2 gm.; $MgSO_4$, 3 gm., and dextrose, 10 gm., made up to 10.5 liters with distilled water.

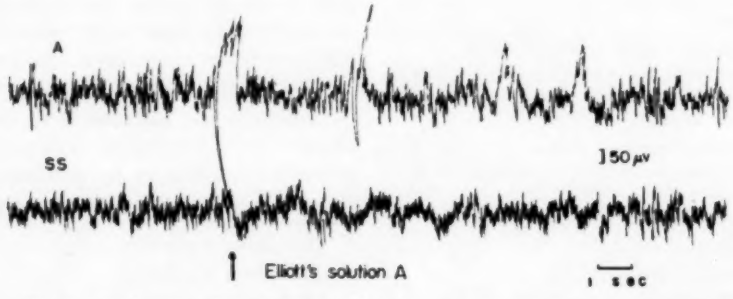

Fig. 1.—Excitatory effect of Elliott's Solution A when sprayed on the cortex.

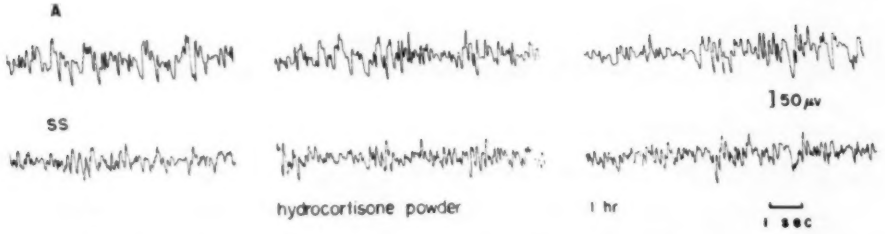

Fig. 2.—Normal ECG, unaltered by local application of hydrocortisone acetate in powder.

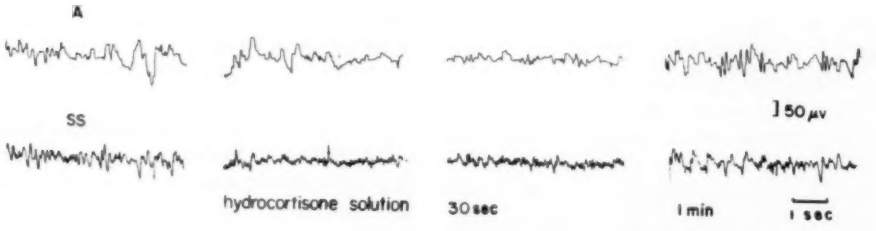

Fig. 3.—Spray of hydrocortisone in Elliott's Solution A. Brief excitatory effect at 30 seconds and normal trace at 1 minute.

sone was administered in solution form, a brief excitatory effect, similar to that of Elliott's Solution A or saline, occurred, lasting approximately 30 seconds, with complete return to the resting state (Fig. 3). It was our impression that the mechanical effect of the spray striking pial vessels was sufficient to cause this brief effect and that it was not related to the hydrocortisone per se.

Nicotinamide was administered topically in the same manner but showed strikingly different effects. After normal base lines had been obtained, nicotinamide in powder form was applied to the cortex between the two recording electrodes. After a latency of several seconds, a marked excitatory effect was observed; increase in frequency and decrease in amplitude were noted, which persisted virtually unchanged for 7 to 15 minutes in various experiments (Fig. 4). It was reversed after a latency of several seconds by washing the cortex with Elliott's Solution A.

Nicotinamide spray had the same effect on cortical electrical activity but without the latent period observed with the powder (Fig. 5).

In one experiment there was an apparent "protective" effect of hydrocortisone on nicotinamide-induced changes. When hydrocortisone and nicotinamide solution was sprayed on the cortex, the excitatory effect was not seen, except for occasional transient "blocking" (Fig. 5). To examine further the possibility of a protective effect of hydrocortisone,

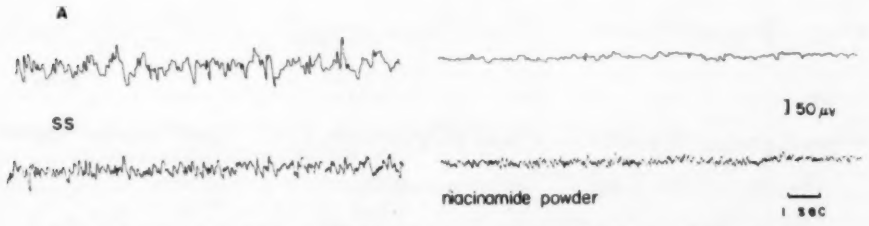

Fig. 4.—Increase in frequency of ECG as result of topical application of nicotinamide powder.

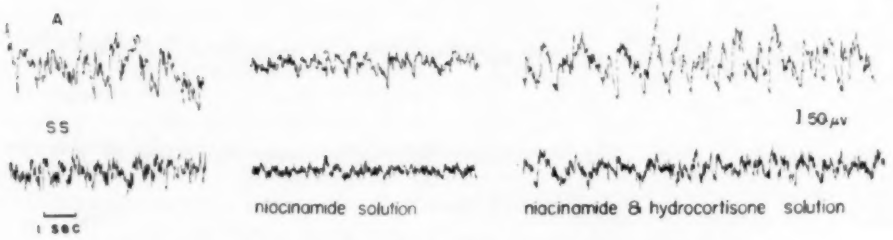

Fig. 5.—Normal trace to compare with the effect of spray of 5% nicotinamide solution. "Protective" action of the hydrocortisone is shown on the left.

the following experiments were carried out in the animal that had 30 mg. of hydrocortisone powder in the cortex for four days. Figure 6 shows the effect of nicotinamide powder applied to the cortical surface. The immediate change was less marked than in animals without treatment, but after 30 seconds a definite excitation occurred. The "protective" effect, therefore, was not so apparent under these circumstances, but it appeared to delay the onset of nicotinamide-induced fast activity.

There was no evidence of high-voltage epileptiform discharge in these experiments.

(*b*) Effect of Strychnine-Induced Spikes: Neither hydrocortisone nor nicotinamide had any appreciable effect on strychnine-induced spike discharges.

(*c*) Evoked Potentials: The wave forms, amplitude, and localization of evoked potentials to auditory and tactile stimuli (clicks and touch to paw) were observed before, during, and after topical application of nicotinamide and hydrocortisone in powder and solution forms. There was no evidence of any alteration. In addition, the "arousal" effect of tactile or auditory stimuli was unchanged by the drugs.

II. Changes in the Vessels

There were no vessel changes when hydrocortisone was applied to the cortex in powder and in solution alone, and when the drug was combined with nicotinamide, the vasodilatation of the cortical vessels was slightly diminished.

Vasodilatation of small capillaries occurred when nicotinamide in powder and in solution was applied to the cortex.

III. Pathology

Pyknosis of a few cortical neurones was the only change found in 6 of the 10 animals. There were no acute inflammatory changes in the vessels.

COMMENT

There is general agreement that corticotropin and adrenal cortical steroids, when given parenterally, affect the electrical activity of the brain, though the mechanism of action is still debated. When hydrocortisone was applied topically to the cortex, there were no changes in the ECG, in the acute

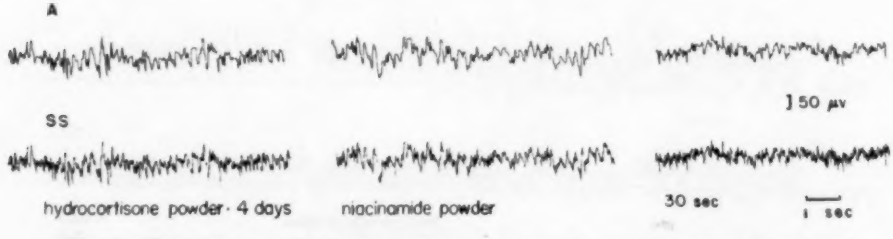

Fig. 6.—Normal trace of ECG when hydrocortisone acetate powder has been four days in the cortex. Application of nicotinamide powder causes a delayed excitatory effect at 30 seconds.

experiments, or when the hormone had been four days in the cortex, thus indicating no direct action of the hormone on the cortical electrical activity of the brain. That there were no changes in the ECG cannot be attributed to a low solubility of the hormone, for there were no changes in the ECG of the four-day animal, when diffusion of the hormone had taken place.

Nicotinamide produces less marked vasodilatation than does nicotinic acid. The latter drug has received attention lately by neurologists and psychiatrists because of its effects on the treatment of schizophrenia. Nicotinic acid (oral or intravenous) produces slight increase in frequency of the EEG in normal subjects.[22] In the present experiment the local application of nicotinamide to the cortex produces a similar increase in frequency in the ECG. The action of nicotinic acid has been attributed to a direct participation, as co-enzyme, in the carbohydrate metabolism of the brain.[22] Chemical studies made by Kety [23] of brain circulation showed no alterations in oxygen metabolism, nor were there any changes in brain vessels. When nicotinamide is applied locally to the cortex, vessel changes occur, and, as a consequence, changes in circulation. The action may be due to alteration in oxygen consumption. It is also possible that the vessel changes may be due to the change in electrical pattern, if the latter is a result of increase in neuronal firing, requiring an increased oxygen supply.

Topical hydrocortisone "blocks" the effects of nicotinamide on the ECG, has no effect on the vessels, and diminishes the degree of vasodilatation caused by nicotinamide. The protective action of local desoxycorticosterone on the vasodilatation of the cortical vessels should be mentioned.[24]

These experiments show that changes in the electrical activity of the brain caused by hydrocortisone directly applied to the cortex are minimal. The hormone appears to exert a protective homeostatic action on the electrical activity of the brain cortex when nicotinamide is applied locally, in powder and in solution.

SUMMARY

Local application of hydrocortisone acetate, in powder or in solution, does not produce changes in the electrocorticogram in cats and blocks transitorily the changes (increase in frequency and decrease in amplitude of the ECG) induced by local application of nicotinamide. Topical nicotinamide causes vasodilatation of cortical vessels, whereas hydrocortisone has no effect on the vessels.

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Neuritis Associated with Systemic Lupus Erythematosus

A Report of Five Cases, with Necropsy in Two

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Recent studies * have indicated that permanent or transient involvement of any part of the nervous system may occur at any time during the course of systemic lupus erythematosus. While disorders of peripheral nerves might be expected to take place in association with this generalized disease, few reports have appeared in the literature suggesting that this actually happens.

Sedgwick and Von Hagan,[4] in 1948, noted one case of lupus erythematosus showing mild mixed polyneuritis and increased total protein in the cerebrospinal fluid. Heptinstall and Sowry,[5] in 1952, reported a case in which examination revealed the neuritic involvement to be rather symmetrical and the total protein in the cerebrospinal fluid to be elevated. Pathologic examination revealed patchy loss of the nerve fibers in the peripheral nerves with variable thickening of the endoneurium. One blood vessel of the epineurium was illustrated, showing focal areas of occlusion with recanalization and, in relationship to the arteriole, chronic inflammatory cells. Eccentric intimal thickening, but with preservation of the internal elastic membrane, was observed in other vessels. No vascular lesions were present in the brain. Typical wire-loop lesions were present in the kidneys, as well as patchy necrosis of some of the glomeruli with crescent formation, and concentric vascular thickening of the splenic arteries. The authors concluded that the peripheral neuritis was due to ischemia resulting from vascular lesions in the vessels supplying the nerves, similar in nature to that described in polyarteritis.

Since 1949 five patients who had systemic lupus erythematosus with associated neuritic involvement have been examined clinically at the Mayo Clinic. In two of these cases necropsy studies were made. Because of the paucity of reports of such cases in the literature, it seemed of interest to present these cases.

REPORT OF CASES

CASE 1.—A girl, aged 13, first admitted to the hospital on Aug. 18, 1950, complained of a "sunburn" over her cheeks and nose, anorexia, and fever during the week before examination. At the time of onset of the fever she also noticed some numbness and "tightness" of her hands and transient migrating pains in the knees and shoulders.

General examination revealed an acutely ill girl with a temperature of 102 F by mouth. The pulse rate was 100 beats per minute, and the respiratory rate was 20 per minute. The blood pressure was 130 mm. of mercury systolic and 68 mm. diastolic. A fiery-red "butterfly" rash was present over the nose and cheeks in the distribution typical of that seen in lupus erythematosus. The liver was palpable. Neurologic examination revealed moderate to marked weakness of the muscles of the extremities with more pronounced weakness in the proximal muscle groups than in those located distally in the extremities. Choreiform movements of the arms

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* References 1-3.

and legs were seen from time to time. The deep muscle stretch reflexes were absent throughout. The plantar responses were normal. Sensory examination demonstrated a rather marked reduction of position sense of the fingers and toes, but its results were otherwise within the limits of normal. The patient was rather labile emotionally.

Laboratory examinations revealed 3000 leucocytes per cubic millimeter. The sedimentation rate was 89 mm. in one hour (Westergren). The blood Kline reaction was doubtful, and the blood Kahn reaction was 3+. The hemoglobin value, erythrocyte count, and results of urinalysis were normal. Sternal-aspiration material was found to contain typical L.E. cells. Examination of cerebrospinal fluid revealed a total protein of 240 mg. per 100 cc., but its results were otherwise normal.

Because of the typical erythema of the face, the joint pains, fever, palpable liver, signs of involvement of the nervous system, leucopenia, elevated sedimentation rate, positive blood Kahn reaction, and the L.E. cells found in the sternal marrow, the diagnosis of systemic lupus erythematosus was made.

Over the next two months, while cortisone was being given, the patient improved somewhat, and she was dismissed to go home in October, 1950. The improvement continued until December, 1950, when fever again developed. Her condition worsened progressively, and she was readmitted to the hospital on Feb. 5, 1951. At this time the results of general and laboratory examinations were similar to those obtained during her first admission.

The neurologic examination revealed intermittent diplopia, nystagmus on lateral gaze, and bilateral intention tremor of the hands. Position sense of the fingers and toes continued to be decreased, and the vibratory sense, stereognosis, and light touch were impaired in the hands and feet. There was hypalgesia to pinprick over the distribution of the left fifth cranial nerve. There was some atrophy of the muscles of the extremities, particularly the small muscles of the hands, with rather diffuse muscle weakness, now more pronounced in the distal muscles. The deep muscle stretch reflexes remained absent. There was limitation of range of motion of the knee and ankle joints.

During the patient's second period of observation in the hospital, although given a course of cortisone, she became progressively worse, ran an intermittent fever, and died Feb. 27, 1951.

At necropsy, the results of gross examination of the brain, spinal cord, and peripheral nerves were essentially normal.

Histologic examination of the lumbosacral nerves revealed a diffuse degeneration of the axis cylinders (Fig. 1*a*). Many were missing; others showed distortion and fragmentation, with many curled segments, resembling Arabic writing. The myelin-sheath stains showed a loss of myelin corresponding to the loss of axis cylinders. Prominent were focal areas of necrosis of the walls of the small arteries (Fig. 1*b*) of the nerves, consisting of fibrin exudation within the walls and superimposed thrombi, and a diffuse infiltration by neutrophiles both within and around the arterial walls. The vascular inflammation could not be directly related morphologically to the areas of degeneration in the nerves. Sections of the Gasserian ganglion showed similar vascular degeneration and inflammation, but, in addition, there was a diffuse degeneration of the ganglion cells, with dropping out of many cells and a diffuse proliferation of the capsular cells. Many of the spinal ganglion neurons were degenerating, and the axis cylinders of the spinal ganglion cells showed marked fragmentation, while the neighboring motor root appeared normal.

In sections of the spinal cord the posterior columns throughout showed almost complete degeneration (Fig. 1*c*), consisting of a decrease in the axis cylinders, almost complete loss of the myelin sheaths, and a filling of the sheaths with fat-laden macrophages, while there were numerous swollen astrocytes within the columns. The neurons within the cord appeared essentially normal.

A section through the medulla showed similar vascular degeneration of two portions of the left vertebral artery. Related to this, there was vacuolation of the left medial longitudinal fasciculus and of the left restiform body. The histopathologic findings of the cerebral cortex were limited to a diffuse thickening of the walls of the small arteries, particularly of those in the cortex. This thickening consisted primarily of an increase in nuclei and some increase in the media. There was a mild, diffuse neuronal degeneration of nonspecific character.

There was a diffuse and focal glomerulonephritis, but without "wire looping." Other lesions, resembling those of periarteritis nodosa, were present in the coronary, renal, and mesenteric arteries.

Case 2.—A woman, aged 21, was first admitted to the hospital in September, 1948, because of migrating joint pains, generalized weakness, and intermittent fever of one and a half years' duration, and the history of an erythematous rash over her nose and cheeks a year before. At that time her local physician had diagnosed her illness as lupus erythematosus and had given her bismuth and gold preparations. The erythematous rash cleared in about five weeks, and she improved generally. For several months she had noticed no joint difficulties.

Physical examination disclosed a blood pressure of 110 mm. of mercury systolic and 70 mm. diastolic; the pulse rate was 90 beats per minute; the oral temperature was 98.6 F. Examination of the skin revealed a dusky redness of the lower lids of both eyes. The deep muscle stretch reflexes of both

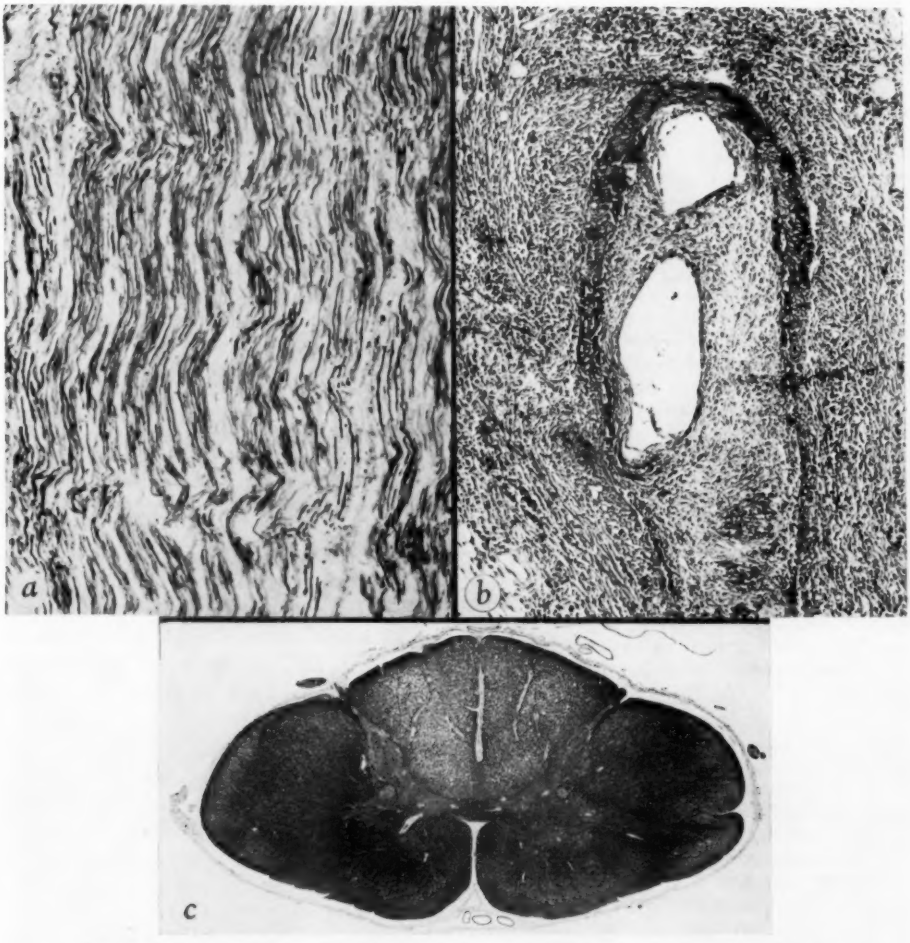

Fig. 1.—(*a*) Sciatic nerve, loss of axis cylinders; Bodian stain; reduced to 97% of mag. × 90. (*b*) Perineural artery, periarteritis nodosa; hematoxylin and eosin; reduced to 97% of mag. × 85. (*c*) Cervical cord, degeneration of posterior columns; Weigert stain; reduced to 97% of mag. × 7.

lower extremities were hypoactive. Otherwise the examination was unrevealing.

Roentgenograms of the thorax disclosed nothing abnormal. The value for hemoglobin was 11.1 gm. per 100 cc. of blood; erythrocytes numbered 3,380,000, and leucocytes 1600, per cubic millimeter of blood. Microscopic examination of a smear of the peripheral blood revealed nothing abnormal. The reaction to the Wassermann test was weakly positive; that to the Hinton test was positive, to the Kahn test 4+, to the Kline test 2+. The value for total proteins was 7.1 mg. per 100 cc. of serum. The albumin-globulin ratio was 4.0 : 3.0. The sedimentation rate was 105 mm. in one hour (Westergren); microscopic examination of sternal-aspiration material revealed L.E. cells.

Because of the history of migratory joint pains, fever, and erythematous rash over the face, as well as the findings of leucopenia, elevated sedimentation rate, blood serologic reactions falsely positive for syphilis, and L.E. cells found in the sternal marrow, it seemed apparent that the patient was suffering from systemic lupus erythematosus.

During the patient's stay in the hospital she was afebrile and she had no exacerbation of symptoms. However, during 1948 and 1949, after returning home, she continued to experience episodes of fever and joint difficulties, and she remained in bed.

At the time of the patient's second admission, in August, 1949, an erythematous rash was noted in the nasolabial region. The results of laboratory studies were similar to those found in 1948. During the latter part of August, 1949, fever developed, with hallucinations, vomiting, and hiccup. About this time she began to complain of tingling of her finger tips, toes and feet, and the tip of her tongue. Neurologic examination revealed absence of the deep muscle stretch reflexes throughout. The proximal muscles of the extremities were found to be very weak, while those toward the periphery of the arms and legs were less so. Two-point discrimination and stereognosis were reduced on the hands, but the results of sensory examination were otherwise normal. Because of the evidence suggesting polyneuritis, examination of the cerebrospinal fluid was done. The total protein in the cerebrospinal fluid was 150 mg. per 100 cc. of fluid; 3 lymphocytes were present in 1 cu. mm.; the Kolmer reaction of the cerebrospinal fluid was negative, and the gold curve was 1343210000.

After the patient's return home, during the remainder of 1949 and the first part of 1950, her illness remained quiescent. However, exacerbations of some of her general symptoms began during the summer and fall of 1950. The weakness of, and paresthesias in, her extremities increased in severity until January, 1950, when improvement began to take place.

At the time of the patient's last admission, in January, 1951, because of increasing fever and joint pain, the neurologic examination revealed improvement of muscle strength. However, sensory studies showed decreased to absent vibratory sensation and hyperalgesia on the lower legs and feet. The deep muscle stretch reflexes continued to be absent. On Jan. 22, 1951, cortisone therapy was begun, but the patient's condition worsened rapidly. Gross hematuria developed, and the platelet count was found to be 35,000 per cubic millimeter of blood. L.E. cells were again found in the sternal-aspiration material. Cortisone therapy was discontinued on Feb. 2, 1951. On Feb. 16, the patient began to have convulsive seizures, and she died on Feb. 19, 1951.

At necropsy the brain, spinal cord, and peripheral nerves appeared grossly normal. Histologic examination of the peripheral nerves, primarily the sciatic and peroneal nerves, revealed that many axis cylinders were missing and others broken, distorted, and twisted (Fig. 2*a*). There was a corresponding loss of myelin sheaths. The blood vessels were slightly thicker than normal, but otherwise revealed no specific change. Sections of the spinal ganglia of the lumbar region showed a diffuse degeneration of many of the ganglion cells with proliferation of the capsular cells (Fig. 2*b*). With silver stains there was a corresponding degeneration of axis cylinders, similar to that in the peripheral nerves. The motor roots did not appear affected. Sections of the spinal cord showed in the lumbar area a complete loss of myelin in the posterior columns (Fig. 2*c*), with replacement by swollen macrophages containing fat, while between the empty sheaths were numerous swollen astrocytes (Fig. 2*d*). The spinal nerve cells appeared essentially normal. Sections of the thoracic and cervical portions of the spinal cord showed the degeneration limited to the fasciculus gracilis and mesial portion of the fasciculus cuneatus.

Multiple sections of the brain showed scattered, but infrequent, focal areas of degeneration, circular in form, consisting of, in some areas, a diffuse loss of normal cells with replacement gliosis, in others, a focal area of acute degeneration with swollen axis cylinders. These areas appeared related to thick-walled blood vessels, with a proliferation of the nuclei of the media. There was no evidence of thrombi, and no mesenchymal tissue reaction was present. In addition, there was a mild nonspecific degeneration of the neurons and some swelling of the oligodendrocytes.

Comment (Cases 1 and 2).—In these two cases there was clinical evidence of widespread involvement of the central nervous system. In addition, however, examination of both revealed evidence of polyneuritis in the form of absent deep muscle stretch reflexes; muscle weakness, more pronounced in the proximal than the distal muscles of the extremities, and decrease of some of those sensations carried in the posterior columns of the spinal cord. These neurologic signs, combined with the low cell counts and elevated total protein determinations found in the cerebrospinal fluid, are consistent with the syndrome of neuronitis (Guillain-Barré). Examination of Case 1 at a later date revealed, in addition, decreased sensation over the distribution of the left fifth cranial nerve.

Pathologic study of Case 1 revealed that the primary lesion appeared to be a degeneration of the posterior root spinal ganglia, with secondary diffuse degeneration of the posterior columns of the whole spinal cord and peripheral degeneration of the spinal nerves. In addition, there were widespread lesions of periarteritis nodosa in the peripheral nerves, and associated with focal infarction of the medulla. In Case 2 there was a similar degeneration of the dorsal root ganglia, primarily

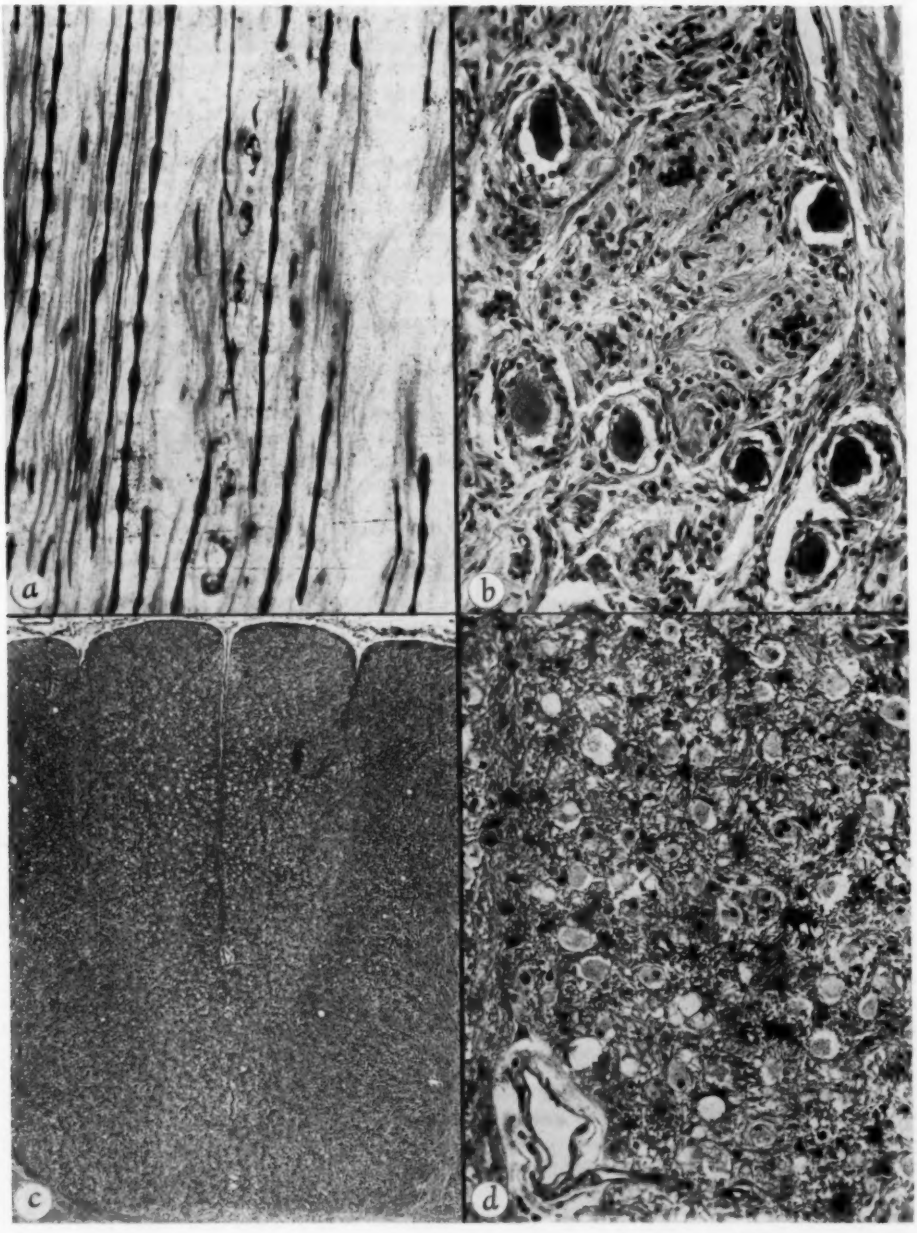

Fig. 2.—(*a*) Sciatic nerve, degeneration of axis cylinder; Bodian stain; reduced to 97% of mag. × 400. (*b*) Posterior root ganglia, degeneration of ganglion cells; hematoxylin and eosin; reduced to 97% of mag. × 200. (*c*) Posterior columns, degeneration of medial tracts; hematoxylin and eosin; reduced to 97% of mag. × 30. (*d*) Posterior columns, secondary degeneration of myelin; hematoxylin and eosin; reduced to 97% of mag. × 200.

in the lumbar region, with a diffuse degeneration of the sensory nerves and of the posterior columns of the spinal cord; but there was no evidence of any primary vascular necrosis. In addition, there were focal areas of necrosis, old and recent, of the brain, apparently perivascular and related to the thickened blood vessels.

In both cases there was a diffuse degeneration of the cortical neurons of nonspecific character, probably terminal in origin. The blood vessels of the nervous system were found to be thickened, a condition similar to that seen in the renal lesions of lupus erythematosus, the so-called wire loops.

Case 3.—In August, 1947, a woman, aged 32, was admitted to the clinic complaining of transient migrating joint pains, which she had experienced for 20 years. For the past year she had suffered from increased difficulty with her joints and had had three episodes of pleurisy. In April, 1947, a vesicular eruption had been noted over the right side of the chest, which was diagnosed as being herpes zoster. About May, 1947, the chest and joint pains increased in severity and fever developed. She also noticed a feeling of generalized weakness, as well as numbness and tingling of both legs and feet and intermittent diplopia. By July, 1947, the weakness had become so severe that she was hospitalized.

The patient stated that while in the hospital she had been given four blood transfusions because of anemia. In addition, she had received five "gold shots," for reasons not known to her. After the hospitalization her symptoms gradually subsided.

Physical examination disclosed a blood pressure of 94 mm. of mercury systolic and 70 mm. diastolic; the pulse rate was 96 per minute, and the temperature was 99.4 F. Slight nonpitting edema was present at both ankles. Only minimal scattered joint changes were found. Cutaneous lesions were not seen, and the results of the remainder of the general examination were normal. Sensation was found to be intact except for a strip of hypalgesia over the distribution of the right third thoracic spinal nerve, where the herpes zoster had been located. The calves of the patient's legs were moderately tender to palpation. The deep muscle stretch reflexes of both lower extremities were markedly reduced. Plantar extension of the left foot was very weak and that of the right foot slightly so. Sensation was normal throughout.

Roentgenograms of the thorax disclosed nothing abnormal. Those of the hands revealed slight periarticular swelling of several of the phalangeal joints. The value for hemoglobin was 10.5 gm. per 100 cc. of blood; erythrocytes numbered 4,100,000, and leucocytes 3700, per cubic millimeter of blood. The sedimentation rate was 110 mm. in one hour (Westergren). Reaction to the Kline test was negative; the value for total protein was 6.9 mg. per 100 cc. of serum; the albumin-globulin ratio was 3.67 : 2.81. The results of urinalysis, including urine studies for arsenic, lead, and porphyrins, were normal. Analysis of gastric contents showed free hydrochloric acid. Sternal aspiration revealed typical L.E. cells.

Because of the history of transient joint pains and three episodes of pleurisy, evidence of disease of the nervous system, leucopenia, elevated sedimentation rate, and L.E. cells found in the sternal marrow, it was felt that the patient was suffering from systemic lupus erythematosus.

When last seen, on June 12, 1953, the patient stated that any exertion on her part or exposure to the sun would cause her to suffer extreme weakness and muscle, joint, and substernal pains. These symptoms would usually cause her to go to bed for several days. The results of physical and neurologic examination were unchanged from those of 1947 except that the rheumatoid joint changes were more prominent in her fingers and the anterior tibial muscles were stronger. Her wrists and elbow joints were moderately tender to forced motion. The results of laboratory studies were unchanged. The sedimentation rate was 83 mm. in one hour (Westergren). Reaction to the Kline test remained negative. The reaction to the L.E. clot test was moderately positive. On microscopic examination, a specimen removed from the left trapezius muscle was normal.

Comment (Case 3).—In this case the only clinical evidence of involvement of the nervous system associated with the systemic lupus erythematosus was the decreased deep muscle stretch reflexes of the lower extremities and weakness of plantar extension of both feet. In contradistinction to Cases 1 and 2, the involvement of the peripheral nerves appeared to be motor and distal in type.

Case 4.—The patient, a white single woman, aged 18, was first admitted to the hospital on Sept. 19, 1953, complaining that she had suffered for five months from intermittent fever, swelling of the periorbital tissues of both eyes, generalized bodily soreness, transient swelling and pain of the finger joints and left wrist, an episode of an increased number of leucocytes and erythrocytes in her cerebrospinal fluid, which her local physician had diagnosed as meningitis, as well as mental confusion, and recently tingling in her arms and legs.

On physical examination the patient appeared chronically ill. The anterior cervical, axillary, and supratrochlear lymph nodes were enlarged. The

posterior pharyngeal wall was injected. There was slight flexure contraction of the knee joints. The blood pressure was 100 mm. of mercury systolic and 80 mm. diastolic; the pulse rate was 108 per minute, and the temperature was 99.6 F. Neurologic examination revealed tenderness of muscles on palpation of the arms and legs. There was evidence of minimal weakness of the muscles of the right seventh cranial nerve, indicating involvement of the right seventh cranial nerve nucleus or peripheral fibers. While all muscle groups of the extremities seemed weaker than might have been expected, the triceps muscles seemed weaker than the rest. The deep muscle stretch reflexes were normal. Sensory examination revealed decreased pain, light touch, and vibratory sensation of the feet and hands and lower segments of the legs and arms in a "stocking" and "glove" distribution. Position sense of the toes was also mildly impaired. In addition, there was mild hypalgesia over the distribution of the left fifth cranial nerve and left second cervical spinal nerve.

Roentgenograms of the thorax disclosed nothing abnormal. The value for hemoglobin was 11 gm. per 100 cc. of blood; erythrocytes numbered 3,670,000, and leucocytes 2800, per cubic millimeter of blood. Examination of a smear of the peripheral blood revealed nothing abnormal. The sedimentation rate was 100 mm. in one hour (Westergren); reaction of the Kline test was negative; the results of urinalysis were normal, and the result of the L.E. clot test was strongly positive. Examination of the cerebrospinal fluid, withdrawn from the lumbar region, revealed the initial pressure to be 12 cm. of cerebrospinal fluid. There were 3 lymphocytes per cubic millimeter. The total protein was 40 mg. per 100 cc. and the sugar 47 mg. per 100 cc.

Because of the intermittent fever, joint difficulties, swelling of the face, evidence of disease of the nervous system, lymphadenopathy, leucopenia, elevation of the sedimentation rate, and a strongly positive result of the L.E. clot test, it seemed apparent that the patient was suffering from systemic lupus erythematosus.

During the patient's stay in the hospital her course was stormy and she was given various antibiotic drugs. As time passed, moderately severe anemia developed, and she was given blood transfusions. Intermittent fever was common. On occasions she lost consciousness for considerable periods and was observed to have generalized tonic muscle spasms with opisthotonos. It was noted that for a short period an erythema developed over the nose which was located so as to suggest a "butterfly" distribution.

However, concurrent with cortisone therapy, given in varying doses, there was gradual improvement of her general state, as well as of the neuritis. After 93 days of hospitalization she was sufficiently recovered to return home. Since that time several mild relapses with fever have occurred, but there has been no recurrence of her neuritic difficulties.

Comment (Case 4).—As in Cases 1 and 2 there was some evidence of involvement of the central nervous system, as well as the peripheral nerves. The mild "stocking" and "glove" diminution of sensation over the hands, lower portion of the legs, and feet suggested a mild symmetrical peripheral neuritis of the sensory type resembling the peripheral motor neuritis found in Case 3. However, the weakness of the muscles of expression of the right side of the face, suggesting peripheral involvement of the seventh cranial nerve or involvement of its nucleus, and the decrease of sensation over the left fifth cranial nerve (as in Case 1) and the second cervical spinal nerve suggest the hit-and-miss involvement characteristic of mononeuritis multiplex or lesions in the respective nuclei.

CASE 5.—A full report of this case has been given by Siekert and Clark.[8] The salient features are as follows:

There were episodes of transient diplopia and dizziness; blurring of vision of the left eye; tingling of the left foot; episodes of vertigo and severe nausea, with subsequent improvement over a period of six months; normal cerebrospinal fluid, except for a total protein content of 100 mg. per 100 cc., and muscle weakness, atrophy, and sensory loss, primarily over the distribution of the right eighth cervical spinal nerve or lower cord of the brachial plexus. Because of the course of the disease (swollen and painful joints, intermittent fever, and evanescent neurologic complaints) and the laboratory findings (leucopenia, elevated sedimentation rate, and a positive result of the L.E. clot test), it was apparent that the patient was suffering from systemic lupus erythematosus.

Comment (Case 5).—In contradistinction to the symmetrical neuritis found in the other cases, this patient presented evidence of a neuritis involving primarily the right eighth cervical spinal nerve or lower cord of the brachial plexus, as well as paresthesias of the left leg, suggesting a mononeuritis or monoradiculitis multiplex type of disorder.

GENERAL COMMENT

The cases presented here for study had variable manifestations of neuritis, in addition to the rather typical signs and symptoms

of systemic lupus erythematosus. There was a history of migrating arthralgias, fever, weakness, and malaise in all cases. Though not considered necessary for the diagnosis of lupus erythematosus, the erythematous rash over the nose and cheeks was present in several cases. Leucopenia, elevation of the sedimentation rate, false-positive blood serologic reactions for syphilis, thrombocytopenia, L. E. cells in sternal-aspiration material, and positive results of L. E. clot tests were found in some or all of the patients. In addition to the neuritis, four of the five patients had other signs of involvement of the nervous system, such as diplopia, nystagmus, confusion, anxiety, and convulsions.

The causes of neuritis and the diseases with which neuritis is associated are multiple. New manifestations of disease are frequently coming to the attention of the physician, and in recent years the "collagen diseases" have come to be recognized as important causes of neuritis and other neurologic disorders. The collagen disease in which neuritis most commonly appears is periarteritis nodosa. In this disorder the clinical features of neuritis have been characterized as mononeuritis multiplex, although cases of symmetrical multiple neuritis have been observed. Other causes of mononeuritis or monoradiculitis are pressure, other vascular lesions, or metastatic neoplastic deposits. The causes of multiple neuritis, especially if the clinical features appear in a symmetrical fashion, may be toxic or metabolic in nature.

The study of five cases of lupus erythematosus reported here reveals no consistent type of neuritic involvement. Examination of Cases 1 and 2 revealed findings suggestive of the syndrome of neuronitis (Guillain-Barré). In Case 3, and to some extent in Case 4, the neuritis seemed to be peripheral in type. However, other findings in Case 4, as well as those in Case 5, suggest involvement of the mononeuritis multiplex type. The reasons for the variation in the intensity and the actual character of these changes are not clear. In Case 1 there was evidence of periarteritis nodosa when the sections of the peripheral nerves were studied histologically, as well as similar changes in the remainder of the body. As this patient received cortisone, it is of interest to note that Slocumb [6] has reported the appearance of periarteritis nodosa in rheumatic patients on withdrawal of cortisone. However, in our Case 1 it cannot be said that cortisone was necessarily related to the arteritis, since vascular changes have been noted in lupus erythematosus before the cortisone era. This was evident in the case reported by Daly.[7]

It is probable that tissue reactions of various types occur in the course of the collagen diseases. That the neurologic changes in Case 1 were not due solely to the lesions of periarteritis nodosa is evident from the advanced degree of degeneration of the posterior columns, which appears to have taken place before the more recent, active lesions of periarteritis nodosa, and, more importantly, from the fact that in Case 2 evidence of degeneration of the posterior ganglia, peripheral nerves, and posterior columns almost identical with those of Case 1 was present without any evidence of periarteritis nodosa. Thus, although Case 1 resembles the case reported by Heptinstall and Sowry [5] in having both peripheral nerve degeneration and periarteritis nodosa, Case 2 differs in the absence of periarteritis nodosa and strongly suggests that the primary lesion may be other than vascular.

In addition to the foregoing clinicopathologic findings, there are some points of anatomic interest. Loss of position sense and loss of vibration sense have sometimes been spoken of as posterior column signs. This is not always accurate, since loss of such forms of sensation may also be due to lesions or disorders of the posterior ganglion cells. In Cases 1 and 2 the marked loss of myelin in the posterior white columns was probably due to secondary degeneration along the axon resulting from the serious changes in the posterior ganglion cells.

SUMMARY

The case histories of five patients with varying forms of neuritis associated with systemic lupus erythematosus are reported.

In two of these cases necropsy revealed focal degeneration of the peripheral nerves and posterior ganglia and secondary degeneration of the posterior columns. In one case, no associated cause for this degeneration was noted; in the other, lesions of periarteritis of the blood vessels of the peripheral nerves and elsewhere were present.

It appears that vascular changes by themselves do not account for the presence of the neuritis in all cases. Examples of mononeuritis and multiple neuritis are reported.

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News and Comment

ANNOUNCEMENTS

The American Academy of Neurological Surgery Award.—The American Academy of Neurological Surgery announces its second annual award. This award is intended as an incentive to research by younger neurosurgeons during or immediately following their formal training. It is given for the best paper submitted reporting original clinical or laboratory research pertaining to the nervous system for which the author has had the principal responsibility. Eligibility is confined to doctors of medicine currently in training in neurological surgery or who have completed their training since June 30, 1954. Those who have been certified by the American Board of Neurological Surgery; are Fellows of the Royal College of Surgeons, Canada, subspecialty neurosurgery, or have other equivalent certification are not eligible. The paper shall not have been accepted for publication in any journal, but may have been submitted as a thesis for a degree. When subsequently published, it shall carry the footnote, "American Academy of Neurological Surgery Award, 1956."

The recipient of the award will be expected to present the paper at the annual meeting of the American Academy of Neurological Surgery in Phoenix, Ariz., Nov. 7-11, 1956. The expenses of the recipient, while attending the meeting and in travel from his home (if living outside the North American Continent from the port of entry into the United States or Canada) will be borne by the Academy. In addition, there will be an honorarium of $100.

Manuscripts for consideration by the committee should be sent to Dr. Edwin B. Boldrey, Chairman of the Award Committee, University of California School of Medicine, San Francisco, and should be submitted before July 15, 1956. A résumé of the candidate's postdoctoral training must accompany the paper when submitted.

Regional Differences in Seizure Susceptibility in Monkey Cortex

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INTRODUCTION

It is generally accepted that the modern era in knowledge concerning the epilepsies began with the writings of Hughlings Jackson.[1] To him, these disorders resulted from the "sudden temporary excessive discharge of some highly unstable region" of the brain leading to "secondary discharge of healthy cells in other centers." He conceptualized regarding the diverse clinical patterns of epileptic phenomena by stating: "According as the seat of the discharging lesion varies the symptoms of the paroxysm vary."

These precepts of Jackson's, predicated upon the abnormal excitability of focally discrete collections of cells, have been expanded tremendously in recent years by the vast experience of Penfield and his associates. Penfield and Jasper state, in speaking of focal epilepsy: "Theoretically, a seizure may originate in any ganglionic part of the central nervous system. However, there are zones of predilection, such as the cerebral cortex, and there are areas that are completely refractory to epileptic discharge in man, such as the cortex of the cerebellum."[2] It is known, additionally, from clinical reports that there are differences in the tendency of various cerebrocortical loci to initiate seizure activity when irritated by a focal lesion.* Moreover, the induction of experimental seizure patterns by alumina (aluminum oxide) cream applied to cortex [5] and by elicitation of local after-discharges on the brain surface [6] likewise reflects regional differences in cortical epileptogenic susceptibility. Yet no systematic study has been undertaken to localize cortical regions displaying hypersusceptibility as contrasted with those relatively resistant to seizure discharge.

Such study requires a procedure which permits rapid assessment of seizure onset and dissemination, either by following the behavioral manifestation induced by epileptogenic lesions in animals or by recording the electrical propagation of locally induced cortical after-discharge. During an earlier survey of corticifugal connections to the brain stem reticular formation, using evoked potential techniques, we had the impression that those cortical regions having such projections were unusually susceptible to after-discharge and other seizure-like activity.[7] Therefore, we decided to apply standardized electrical stimulation to the cortex, in a systematic way, in order to test regional differences relating to the induction of seizure patterns. This method of investigation proved to be particularly useful because it was feasible at each cortical locus to determine the threshold for seizure activation by applying successively more intense stimulation, with other stimulus parameters kept constant. Moreover, it permitted a comparison of the relative excitability of each region tested with

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* References 3 and 4.

other areas similarly stimulated and made it possible to verify responses by subsequent reapplication of the stimulus to each test site.

By this routine of stimulation accompanied by electroencephalographic recording, we examined the entire surface of the cerebral hemisphere in the monkey and found substantial threshold differences relating to the production of seizure activity. The discharge threshold of each area in the cortex of a single animal was found to be remarkably constant when retested at later periods during the experiment, and excitation of comparable regions in different monkeys elicited singularly consistent results. Points within a few millimeters of each other commonly exhibited very different thresholds and quite different channels for spread of seizure activity. The suggestive boundaries defining differences in susceptibility to after-discharge and in the direction of its spread appeared also to be relatively consistent. Whatever may be the mechanisms of the genesis of epilepsy, these data indicate substantial regional differences in cortical susceptibility to the electrical induction of seizures.

METHODS

Experiments were made on a total of eight monkeys. The general procedure in all instances was similar, but craniectomies were varied so as to expose appropriate cortical areas for investigation. In different studies, all surfaces of the entire cerebrum were examined, and each region of brain was analyzed for comparison with other reference loci in at least two animals.

The cortex was exposed and subcortical electrodes placed while the animals were under ether anesthesia, small amounts of procaine being injected into exposure margins and pressure areas. The monkeys were then immobilized with tubocurarine U. S. P. (*d*-tubocurarine) and maintained on artificial respiration. In different experiments, two anterior and two posterior bipolar concentric needle electrodes were placed stereotactically in the following structures: septal nuclei (three monkeys), caudate nucleus (four), globus pallidus (one), corpus callosum (two), fornix (one), amygdala (six), nucleus dorsalis medialis (one), centrum medianum (one), substantia nigra (three), habenular complex (one), decussation of the brachium conjunctivum (one), red nucleus (two), and brain stem reticular formation (five). The location of the electrodes was determined at the conclusion of each experiment by examination of serial sections stained with thionine. Bipolar silver-ball electrodes were used for cortical stimulation and recording, a recording pair invariably being placed for monitoring within 2 mm. of the stimulating electrodes. The cortical recording sites remained fixed throughout each experiment and, except for the monitor pair, were generally located on the convexity. In each instance, sufficient craniectomy exposure was made to permit sampling from a portion of each gross lobe of the brain ipsilaterally and of the sensorimotor region contralaterally. In this manner, it was possible to determine the relationship between activity induced at a stimulus site and the tendency it might have to excite neighborhood discharge in the same lobe, as well as to spread to remote cerebral regions. Recording was made by means of a Grass Model III electroencephalograph. Rectangular pulses maintained at 50-per-second frequency and 5-msec. duration delivered from a Grass stimulator were successively applied to each cortical point in bursts lasting three seconds, with intensities of 5, 10, and 15 volts. If generalized after-discharge occurred with the lower voltages, stronger stimuli were avoided to prevent undue prolongation of cortical recovery. In order that a reasonable constancy of experimental conditions could be maintained, the intervals between stimuli were arbitrarily selected on the basis of return of the EEG tracing to normal.

Because these experiments yielded such a vast amount of raw data, certain criteria were established in order to evaluate the results. These criteria concerned (1) the production of a significant after-discharge at the site of stimulation and (2) the manner in which the discharge was transmitted to other cortical and subcortical regions. If no local after-discharge was excited by the cortical stimulus, the area excited was considered to be a silent one. If induced activity was confined to the monitor, or if it did not extend outside the thalamocortical sector of the stimulus site, the area was considered relatively inactive or capable of exciting only local or restricted firing. If, however, after-discharge initiated locally was recorded diffusely in remote cortical lobes and/or in subcortical structures having widespread projections to the brain, the stimulus site was designated as a highly active epileptogenic focus. Thus, for example, after-discharge evoked in a portion of the frontal lobe, but confined to the stimulus site and to the dorsomedial nucleus of the thalamus, was not considered a diffuse seizure or the point excited an active focus. Constrastingly, activity spreading to reticular formation, centrum medianum of the thalamus, amygdala, septal nuclei, etc., in conjunction with diffuse cortical firing, was so considered.

Because interest was centered primarily on the relative capacity of cortical loci to initiate diffuse seizure discharge, the manner in which such in-

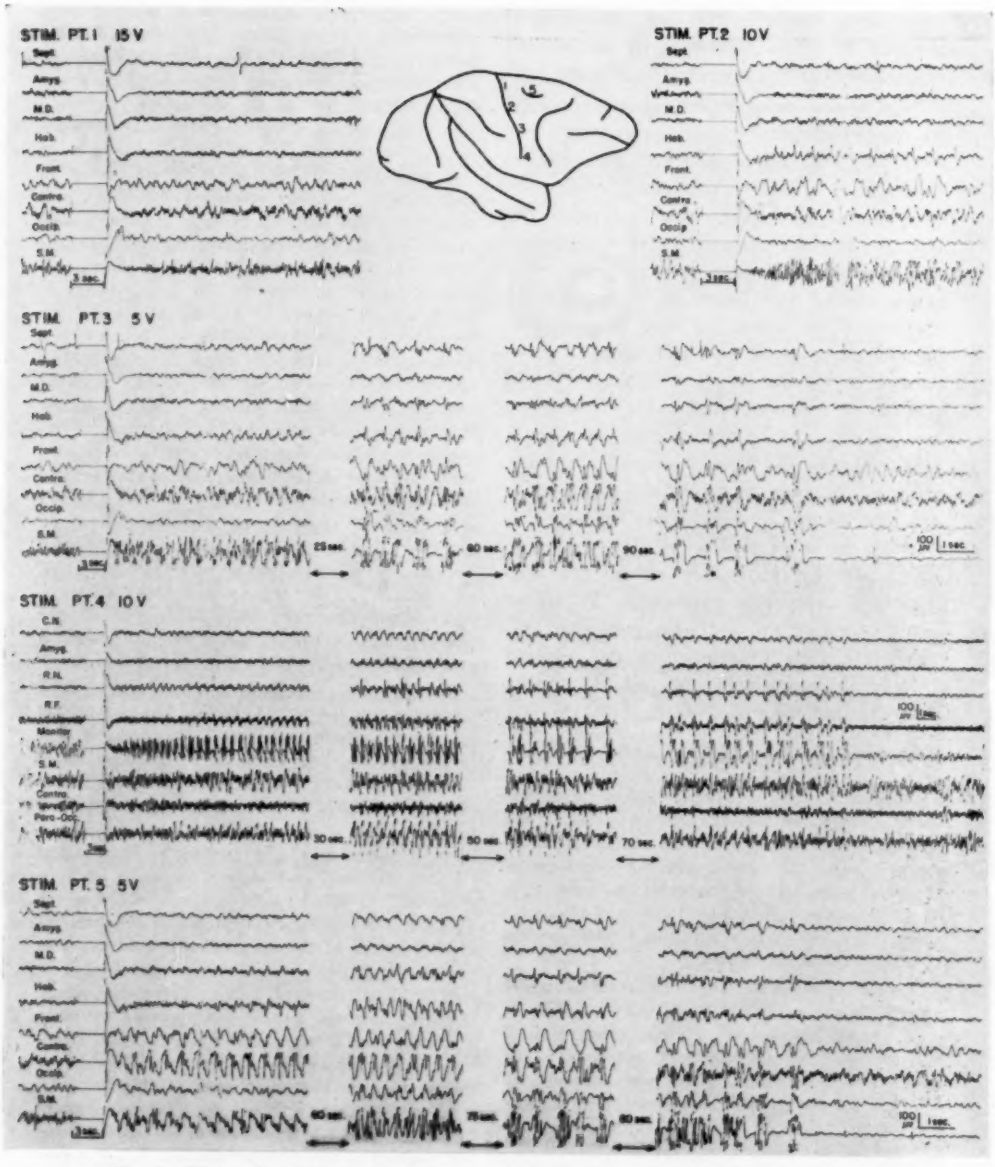

Fig. 1.—Illustrative EEG records indicate variation in the capacity of different points on the frontal lobe (diagram, points *1-5*) to exhibit after-discharge when stimulated directly. High-voltage excitation (15 volts) of point *1* (*Pt. 1*) produced no change in the record and of point *2*, only restricted short-duration firing. Stimulation of points *3*, *4*, and *5*, however, with lower-intensity currents, elicited prolonged and diffuse seizure discharge. The upper four channels in each tracing are brain-stem recordings, while the lower four indicate surface activity. All tracings were recorded continuously, breaks in the illustrated tracing in the lower three lines being indicated by arrows. Abbreviations in this Figure and in Figures 2 and 3: Subcortical recording sites: *Amyg.*, amygdala; *C. M.*, centrum medianum; *C. N.*, caudate nucleus; *Hab.*, habenula; *M. D.*, dorsomedial nucleus of the thalamus; *R. F.*, reticular formation; *R. N.*, red nucleus; *Sept.*, septal nuclei. Cortical recording sites: *Contra.*, contralateral motor region; *Front.*, frontal; *Monitor*, adjacent to stimulus; *Occip.*, occipital; *Para-Occ.*, paraoccipital; *S. M.*, sensorimotor.

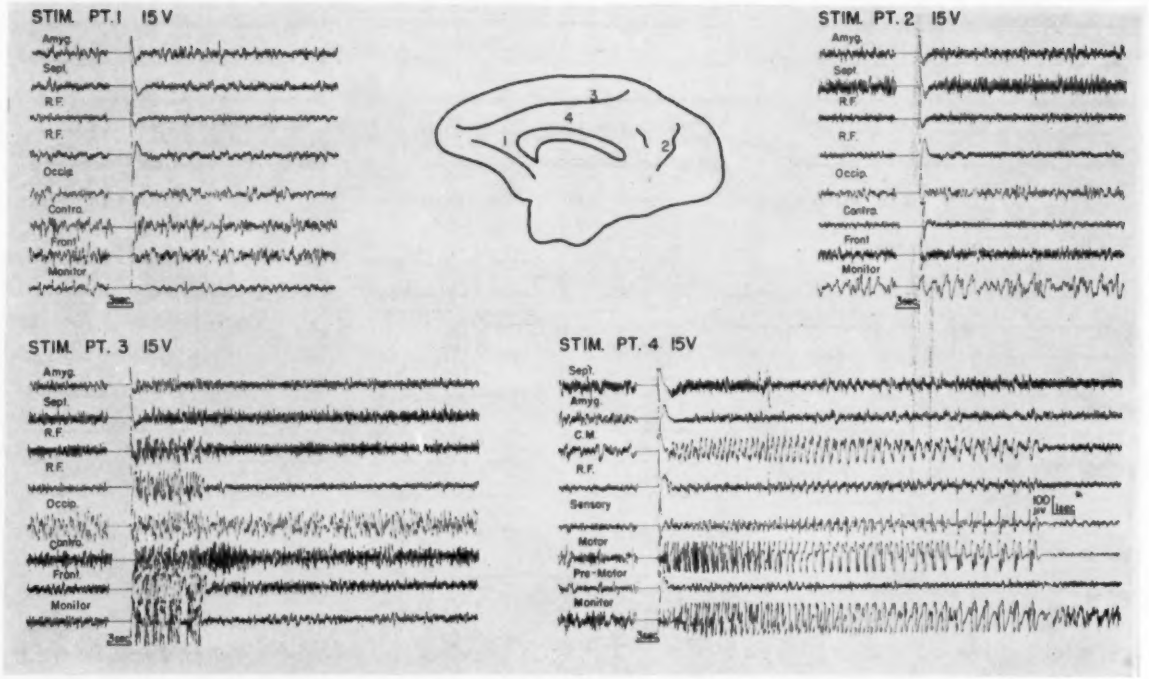

Fig. 2.—The EEG records illustrate differences in excitability of cortical points on the medial surface of the hemisphere. Note diffuse discharge initiated by stimulating points *3* and *4* and absence of firing from points *1* and *2*. For abbreviations, see Figure 1.

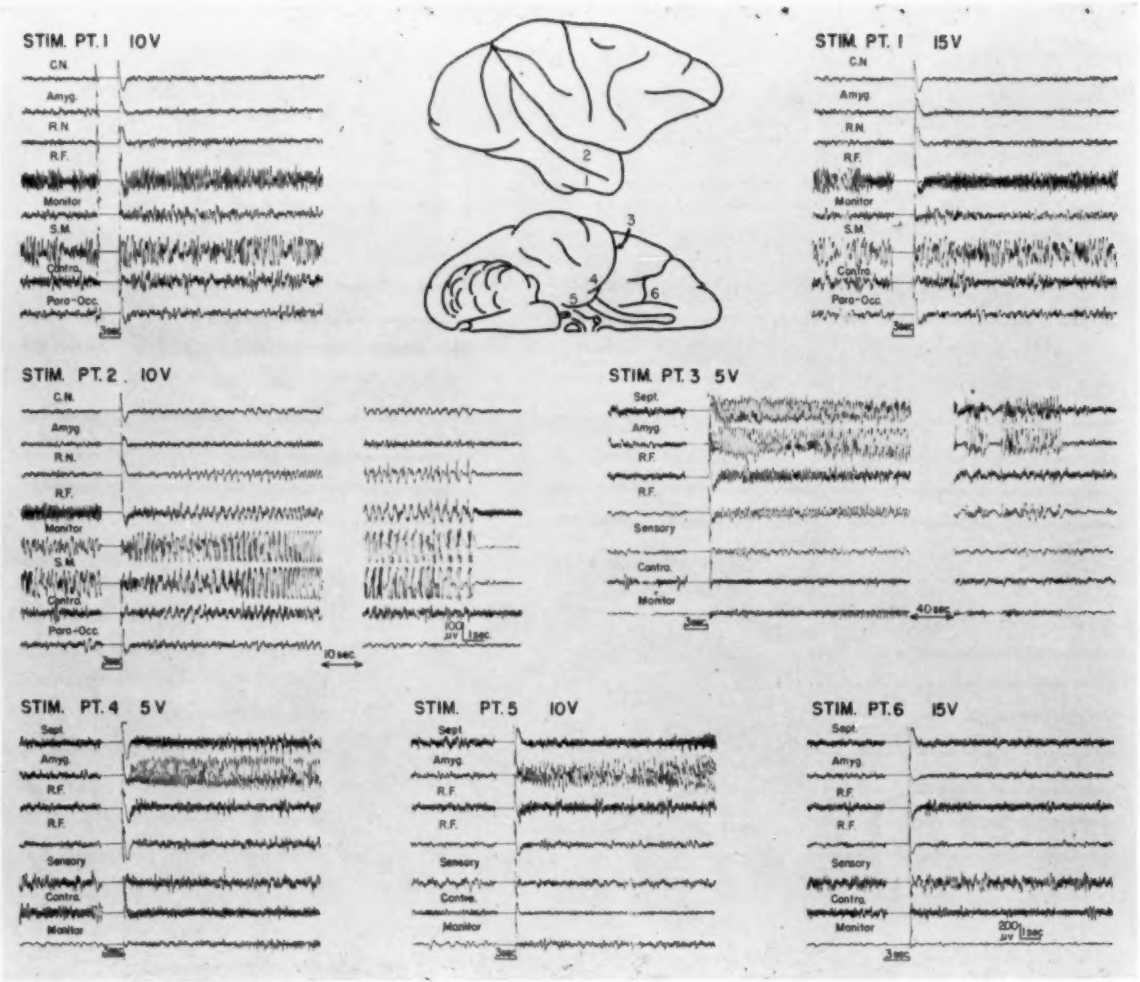

Fig. 3.—The EEG records indicate differences in excitability of cortical points on temporal lobe and undersurface of frontal lobe. Note that records are unchanged when second temporal gyrus and orbital surface are stimulated. Contrastingly, seizure activity is induced by excitation of first temporal gyrus and tip. For abbreviations, see Figure 1.

duced excitation was disseminated was of secondary interest in the study. During the investigations, however, some data were obtained concerning the selective pathways and rates of spread of seizure patterns and the order in which various structures became involved.

RESULTS

Various Patterns Induced by Cortical Stimulation

The first three Figures demonstrate typical tracings, which indicate that appreciable differences in threshold of stimulation are required to elicit after-discharge from various cortical regions. It is evident from Figure 1 that these differences occurred even within a given division of the brain. The application of 15 volts to point *1*, near the vertex in the motor cortex, yielded no after-discharge, whereas 10 volts applied to point *2* gave a short-lived after-discharge which appeared locally, in the ipsilateral frontal lobe and in the brain stem. Five volts delivered to point *3* induced a response in two cortical leads which became generalized after a short delay. Ten volts applied to point *4* elicited a rapidly developing local after-discharge with concurrent appearance of activity in the red nucleus and subsequent generalized dissemination. Five volts applied to point *5* in the premotor cortex was sufficient to induce a discharge which became widely diffused after a short delay. It was also evident that, although different regions might exhibit discharge activity one after the other, the generalized discharges ultimately became synchronous in all leads and tended to terminate simultaneously.

Figure 2 shows responses from the medial surface of the hemisphere and illustrates four patterns indicative of regional differences in cortical susceptibility to after-discharge. Fifteen volts applied to points *1* and *2* on the medial frontal and medial occipital surfaces did not alter the prestimulus tracing. An identical stimulus applied to point *3* yielded a short generalized after-discharge, however, and one to point *4* initiated a more prolonged, apparently less generalized seizure.

Stimulation of point *1* (Fig. 3) on the second temporal gyrus, at 10 and 15 volts, elicited no response. Ten-volt stimulation of point *2* on the first temporal gyrus excited a generalized after-discharge of moderate duration, however, while stimulation of point *3* on the anterior surface of the temporal tip, with only 5-volt intensity, gave rise to a generalized seizure. Stimuli delivered to point *4* on the anteromedial surface of the temporal lobe at 5 volts, and to point *5* in the uncus at 10 volts, yielded a brisk after-discharge particularly evident in the amygdala and septum. Contrastingly, as much as 15 volts applied to point *6*, on the orbital surface, elicited no after-discharge.

General Regional Differences Relating to Seizure Susceptibility

1. *Lateral Surface of the Hemisphere.*—(*a*) Frontal Region: Figure 4 illustrates the gross changes encountered on stimulating different regions at successively higher intensities. The front lobe as far back as the frontal eye fields was found to be relatively unresponsive to stimulation at 5 volts. Responses which were induced by this intensity were limited to localized cortical spread and were of short duration. When a current of 10 volts was applied to the same region, those points locally activated by 5 volts displayed more widespread and prolonged responses, lasting more than 10 seconds and involving the contralateral cortex. Occasionally this after-discharge spread to subcortical structures (e. g., reticular formation and centrum medianum), and sometimes the deep responses appeared after an appreciable delay (e. g., in the amygdala), as though by way of secondary extension. At 15 volts, some loci gave rise to spread to more distant regions, and these were made active for longer periods.

Commonly, the amygdala began to discharge after short delay, but its induced activity was of short duration. A very few points, near the superior arcuate and principal sulci, yielded such prolonged and generalized responses at 5 or 10 volts that higher intensities were not employed. Nevertheless, even at 10 and 15 volts, many points in the frontal region, especially near the frontal pole, were unresponsive.

In summary, active loci in the frontal lobe were relatively rare and scattered in distribution. There was some tendency for grouping of such points, however, in a small region between the superior frontal and the principal sulci and near the frontal border of the superior arcuate sulcus.

(*b*) Motor and Premotor Regions: The motor and premotor regions were much more responsive to stimulation than the more anterior parts of the frontal lobe. Even with 5-volt pulses, the lower part of the motor strip yielded generalized cortical and subcortical spread of after-discharge. Here diffuse seizure initiation was immediate, widespread, and prolonged, although some of the subcortical regions began to respond only after a delay. The upper part of the motor and premotor cortex was definitely less responsive to stimulation, only scattered points giving short-duration after-discharge to 5-volt excitations (Figs. 1 and 4). Only in the region of the superior precentral dimple in the premotor cortex was there a congregation of relatively more responsive points. Even at a stimulus level of 10 volts, however, there remained an obvious distinction in responsiveness between the upper and lower parts of the motor cortex. More of the points in the face and hand regions were strongly positive than in the upper third of the precentral gyrus or the lowest opercular region, and stimulation of this active area had more frequently to be limited to low-intensity pulses in order to avoid excessively prolonged seizures. In stimulating the upper third of the precentral gyrus, we could usually use 15 volts with impunity, and several loci in this region were unresponsive even at this intensity.

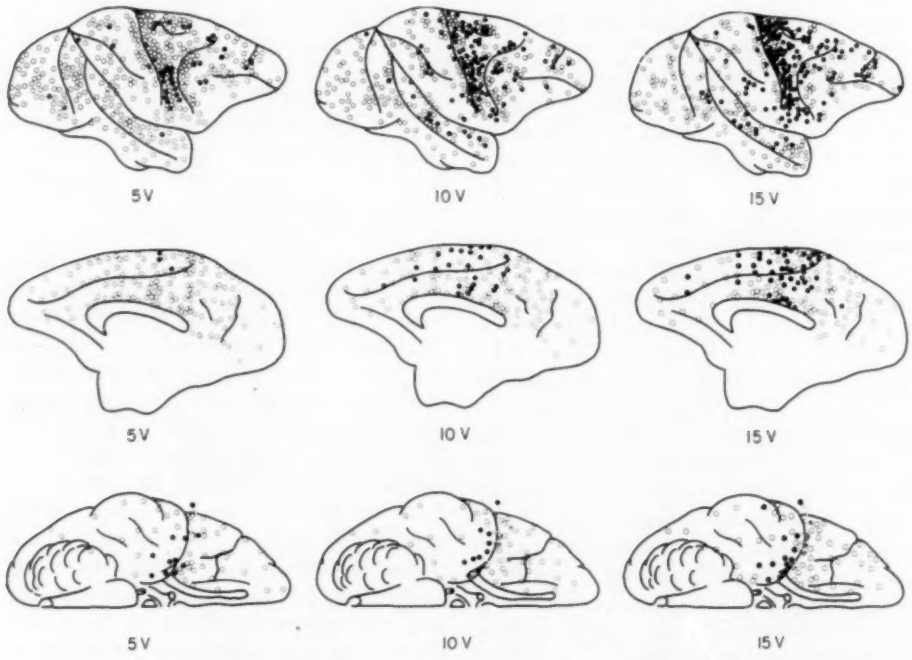

Fig. 4.—All stimulation points are indicated by circles in diagrams of the brain representing lateral (top line), medial (middle line), and inferior (bottom line) surfaces. Filled circles denote seizure induction by stimulation with 5 volts (left column), 10 volts (middle column), and 15 volts (right column), while open circles represent points from which significant after-discharge was not elicited by stimulation with maximum currents employed. See text.

(*c*) Postcentral and Parietal Regions: Behind the Rolandic fissure the cortex was

relatively unresponsive to electrical induction of after-discharge. At excitation intensities of 5 volts, only a few points responded, and these with only brief and localized cortical spread (Figs. 1 and 4). With 10 and 15 volts, only a few isolated points close to the Rolandic fissure yielded brief after-discharge. More posteriorly, in the posterior parietal and paraoccipital region, the response was likewise localized and short-lived, and deep structures displayed either only a brief discharge or a delayed short burst of localized activity. Full-intensity current could be applied to almost all points behind the Rolandic fissure without evoking any generalized after-discharge.

(*d*) The Occipital Region: The entire occipital region was essentially unresponsive at any of the standard intensities of stimulation employed. Only rarely was there even brief activation at the monitor electrodes. It was our practice, after testing a few negative points, to retest some active zone in order to determine whether or not the animal was in good condition. Thereby we made certain, again and again, that the occipital region was normally uniformly unresponsive to 15 or more volts.†

(*e*) The Temporal Lobe: The second temporal gyrus was as unresponsive to electrically evoked after-discharge as was the occipital region. The first temporal gyrus did not respond to 5-volt pulses, but the application of 10 and 15 volts to this area activated amygdaloid and local cortical discharges, which sometimes spread to the contralateral motor cortex (Fig. 3, point *2*). In contrast to loci on the motor and premotor surfaces, where discharges were propagated predominantly to other cortical sites, excitable points in the first temporal gyrus yielded predominantly subcortical spread.

(*f*) General Comment: In brief, the lateral surface of the hemisphere showed the greatest density of responsive points, those most highly active being located in the face and hand portions of the motor cortex. Scattered loci in more anterior parts of the frontal lobe were relatively less responsive and in the postcentral and parietal regions even less so. The superior temporal gyrus did not respond to 5-volt stimulation, but higher intensities applied to this region elecited relatively localized cortical discharge and relatively generalized subcortical spread. Excitation at only 5-volt intensity in the motor area so often yielded widespread and prolonged discharges that higher intensities could not be tested at many points, whereas in the regions of the frontal and occipital poles and the second temporal gyrus, little or no effect could be induced even when as much as 50 volts was applied.

2. *Medial Surface of the Hemisphere.*—On the medial surface of the hemisphere positive results were evoked by stimulation of cortex lying above the central part of the corpus callosum (Fig. 4). The medial surface of the frontal, posterior parietal, and occipital regions were generally unresponsive. Active loci on the medial surface were definitely less excitable than those in the lateral precentral area and were roughly comparable in degree of responsiveness to cortical points on the first temporal convulsion. Activity induced by stimulation by the medial surfaces spread about equally to cortical and subcortical regions.

† A further peculiarity of the occipital region relates to its tendency toward apparently spontaneous, localized, but prolonged, seizure discharges. These occurred rarely near the termination of prolonged experiments, which involved wide exposure of the inferior temporo-occipital region. In such instances cortical and subcortical recording electrodes outside this affected area showed no particular change in activity; it was as though this posterior inferior region of the hemisphere had a seizure circuitry of its own. When such activity died down, it could be readily aroused again by local electrical stimulation but was not usually made active by stimulation elsewhere. We must conclude that this region is capable of a restricted type of seizure pattern, which, when it occurs, may be reinstated by electrical excitation. In contrast to seizure discharge elsewhere, however, this spontaneous temporo-occipital discharge appears to depend upon some locally induced special susceptibility. Moreover, this restricted discharge does not spread to other brain regions in the same manner as that induced by stimulation of other parts of the cortex.

With excitation at 5 volts, no effect was recorded from surface or deep structures as a result of stimulating almost the entire medial surface. Stimulation with 10 and 15 volts applied to active areas yielded immediate responses in both cortical and subcortical recordings which were of short duration (Fig. 2, point *3*), although occasionally subcortical responses appeared after an appreciable delay (Fig. 2, point *4*). In this region it was never necessary to avoid stimulation with the higher voltages. Increasing the intensity from 10 to 15 volts increased the density of responding points within the region where 10 volts gave scattered responses, but did not significantly extend that region. Even 20 to 50 volts applied outside this central part of the medial surface was ineffective in inducing after-discharge.

3. *Inferior Surface of the Hemisphere.*—The orbital surface, the posterior two-thirds of the temporal lobe, and the entire extent of the basal occipital region were essentially unresponsive (Fig. 4). The tip of the temporal lobe on all of its surfaces was exceedingly reactive, however. Responses from the temporal tip, like those from the superior temporal gyrus, tended to spread predominantly subcortically, affecting particularly the amygdala. The anteromedial part of the temporal lobe responded well to 5- and 10-volt stimulation intensities, exhibiting and initiating prolonged and relatively diffuse after-discharge. This region, therefore, appeared to be relatively more responsive than the superior temporal gyrus.

Relationship Between Spread of Seizure Discharge and Activity in Subcortical Structures

As indicated, interest in these experiments centered upon the relative epileptogenetic capacity of different cortical regions, and not upon the manner in which seizure activity induced locally was propagated. In order to determine the degree to which after-discharge became generalized, therefore, it was necessary to record from diverse subcortical structures, as well as from widely separated cortical loci. Our data on subcortical spread of cortically induced seizure discharge hence are insufficient to determine definitively preferential circuits of spread. They do reflect, however, some relative differences in the degree to which some subcortical structures are activated in the course of seizure diffusion.

The reticular formation and septal nuclei appeared to have the highest positive and lowest negative correlation with generalized seizure spread. Discharge was induced in both of these structures by excitation of all active cortical loci in central, frontal, supracallosal, and temporal regions of the brain. Such activity either appeared simultaneously with that excited at the stimulus site (Fig. 3, point *3*; Fig. 2, point *3*) or followed the locally induced discharge by only a brief delay (Fig. 1, points *3* and *4*; Fig. 3, point *2*). Seizure activity in the reticular formation and septum depended upon that in the cortex and always terminated simultaneously with the cortical discharge regardless of the duration of the episode (Figs. 1, 2, and 3).

The reticular formation and septum were definitely more frequently implicated in spread of after-discharge than were the red nucleus and centrum medianum, and these areas, in turn, were involved more commonly than were the caudate nucleus, globus pallidus, dorsomedial nucleus, and amygdala. Generalized spread occurred fairly commonly without involving these latter structures, and on other occasions they became active only after a delay, during which the seizure had become evident elsewhere.

Certain regional differences were observed in the capacity of cortical seizure activity to spread to subcortical structures. Amygdaloid discharge was most effectively induced by temporal lobe stimulation, particularly by excitation of temporal tip and uncus (Fig. 3, points *3*, *4*, *5*). Contrastingly, seizure spread to the amygdala from medial supracallosal (Fig. 2, point *4*), lateral motor and premotor (Fig. 1, point *4*), and frontal (Fig. 1, point *5*) stimulus sites were commonly poorly developed and delayed.

COMMENT

The determination of regional susceptibility differences of cortex to electrical after-discharge excitation in man is difficult because the repeated induction of seizures by stimulation during operation is hazardous and the opportunity to survey the medial and inferior surfaces of the brain adequately is uncommon.[8] Moreover, whatever regional or generalized pathology exists clinically is likely to compromise the data, particularly as epileptogenic lesions for which exploration is made are prone to be extensive. Nevertheless, observations made in man are in no way incompatible with the results in these experiments. Penfield and Rasmussen [8] have observed that seizures can be induced only with great difficulty by stimulating the frontal pole in man but are easily obtained by excitation of more posterior cortical regions. If information obtained during more favorable experimental conditions with monkeys may be extrapolated to man, we would anticipate that a more extensive survey of the medial and inferior surfaces of the human hemisphere as well might reflect comparable differences in seizure susceptibility over the entire hemisphere.

Determining seizure susceptibility in different cortical regions in man by assessing the incidence of epilepsy following pathological brain disturbances is even more difficult. In analyzing data relating to a large number of patients with penetrating cerebral lesions, Russell [9] concluded initially that patients with scars in certain cortical areas were more prone to develop seizures than were those with cicatrix located in other regions. However, he was unable to confirm this hypothesis later.[10] Penfield and Jasper [2] have observed in patients with seizures induced by neoplasms that "the incidence falls as the distance from the sensorimotor cortex increases in any direction," and a somewhat similar observation was reported by Gibbs and Gibbs.[3] Such assessments are difficult because pathological processes—scars, neoplasms, infections, etc.—are usually large, creating uncertainty as to which margin of the lesion impinged upon or induced the epileptogenic area of cortex. Moreover, the nature of the pathological process itself is of at least potential importance in seizure causation. The present experiments are favored by the advantage that the data were determined utilizing normal monkey brains.

Reference has already been made to previous demonstrations of regional differences in the susceptibility of cortex to discharge abnormally upon direct excitation in experimental animals. Rosenblueth and Cannon [11] found that "the greater the distance between any cortical point or area in the same hemisphere and the primary motor point, the more difficult it is to produce tonic-clonic effects in a given muscle, or the less effects of a given stimulus." Gibbs and Gibbs [3] determined that there are threshold differences for stimuli required to produce seizures upon excitation of different cortical and subcortical structures in cats. Segundo, Arana, and French,‡ applying stimulation through chronically implanted electrodes in monkeys, observed that seizures were easily induced by low-voltage excitation of the motor area but could not be initiated by 10 to 20 times as much current delivered to the frontal or occipital poles. It is of interest that the early observations of Bubnoff and Heidenhain [12] and of many that followed § relating to seizures induced by acute or chronic methods were based upon experiments in which the stimuli were applied to the motor area of animals, a region found by us to be the most highly epileptogenic of the entire cortical mantle.

The results of the present experiments show that there are distinct differences from one cortical region to another with respect to threshold for electrically induced, spreading after-discharge (Fig. 5). It appears evident that these differences in excitability are valid physiological expressions of function, and not artifacts attendant upon the experimental method employed. Repeated stimulation of silent areas even at high in-

‡ Segundo, J. P.; Arana, R., and French, J. D.: Behavioral Arousal by Stimulation of the Brain in the Monkey, in preparation.

§ References 5, 13-16.

tensities showed them to remain unresponsive. Repeated and diffuse seizure induction in susceptible regions, however, proved these to retain essentially stable thresholds throughout the experiments. Clearly, local chemical or other environmental changes brought about by the various operative exposures of brain, by the application of electrical stimulation, or by the induced after-discharge itself, acting upon cortical zones otherwise theoretically equally susceptible to seizure induction, cannot operate so consistently as to account for these results. Moreover, such susceptibility differences cannot be imputed to an induced activation of neural structures might subserve their capacity to elicit spreading after-discharge.

The extensive differences in cortical susceptibility are reflected also in differences in subcortical responses. Clearly, some subcortical structures are almost invariably involved in spread of after-discharge induced by cortical stimulation, and, equally clearly, other subcortical regions are rarely so implicated or are implicated only after many other cortical and subcortical regions have been swept into seizure activity. The reticular formation and septal nuclei were found to be the oftenest and earliest involved among the subcortical structures investi-

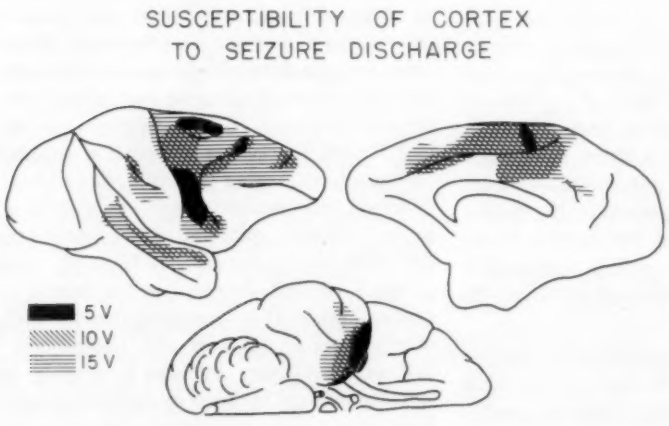

Fig. 5.—The composite results of the experiments are summarized on these diagrams of the brain. The black areas signify that seizures were induced by stimulation with 5 volts, diagonal hatching with 10 volts, and horizontal hatching with 15 volts. See text.

in a certain sequence, as the order for testing and retesting various zones was intentionally varied.

The cortical regions susceptible to electrically induced and disseminating after-discharge do not appear to be related to any obvious or characteristic cytoarchitectural pattern.[17] Even the most highly excitable regions, those occupying the middle two-fifths of the central gyrus and the area just above the superior precentral dimple, exhibit varied microanatomical structures. In fact, a review of all zones found to be relatively susceptible to stimulation suggests that they occupy only portions of various cytoarchitectural fields. Moreover, it is difficult to find obvious similarities in structure common to all of these diverse fields which gated. Other subcortical regions were as commonly, if somewhat less consistently, excited by appropriate cortical stimulation as were these two structures. We found, as others have noted,‖ that the amygdala, while capable of discharging to widely distributed sites of surface stimulation, responded with greatest intensity to excitation of the temporal lobe and related structures. Walker and his associates ¶ indicated that the putamen was most frequently implicated in subcortical spread of seizure activity in-

‖ References 6, 18, and 19.

¶ References 20 and 21. Walker, A. E.; Andy, O. J., and Poggio, S. F.: Role of Basal Ganglia in Convulsions, Program Abstract, 80th Annual Meeting of American Neurological Association, Chicago, June 13-15, 1955.

duced by stimulation of the sensorimotor region, and the caudate nucleus by frontal excitation. No records were obtained from the putamen in the present experiments, but we found that stimulation of the superior temporal gyrus, as well as motor cortex, could induce activation of the caudate nucleus, although, commonly, firing in this structure was appreciably delayed. Moreover, caudate nucleus activation did not occur from many excitation points over the frontal lobe, indicating, perhaps, that this region is not equipotential in its capacity to discharge portions of the basal ganglia. The red nucleus, as noted in three animals, appeared to be relatively more excitable, reflecting perhaps its intimate relation with the reticular formation.

That central brain stem nuclei have widespread anatomical connections capable of effectively disseminating abnormal discharge is well known. Moreover, functions mediated by these structures are among those most critically disturbed during an epileptic fit. The reticular formation [22] and septal nuclei # have been implicated in the arousal response and alerting, as well as in control of motor movement.* Stimulation of the amygdala is known to propagate seizure discharge widely † and to induce episodic states of abnormal behavior or seizures in the animals tested.‡ To all three of these regions have been assigned important visceral and autonomic functions. Thus it is apparent that certain subcortical structures—themselves known to have diffuse connections with other brain stem and cortical areas and to exert widespread influences upon cerebral function—can be secondarily excited to seizure activity by those cortical loci which display persistent after-discharge following direct surface stimulation. Much higher voltages applied to other cortical areas do not excite local after-discharge, nor do they induce seizure activity in the subcortical structures observed here.

References 23 and 24.
* References 25-27.
† References 6, 28, and 29.
‡ References 21 and 30-33.

The significance of the obvious relationship between firing of cortical epileptogenic zones and of discharge induced thereby in the diffusely projecting brain stem structures is not entirely clear. Information concerning connections from some of the cortical regions described to these subcortical areas has been recently reviewed and extended.[7] It was found that frontal occulomotor fields, sensorimotor cortex, paraoccipital region, superior temporal gyrus and tip, orbital surface of frontal lobe and cingulate gyrus connect functionally and anatomically with brain stem structures comprising the reticular activating system. Similar connections have been described between these same cortical areas and the septal nuclei.[34] Direct corticoamygala connections seem to emanate more from rhinencephalic structures [6] and from the temporal lobe,[19] suggesting that the amygdala is primarily susceptible to discharge from stimulating loci in these areas. Yet diffuse reciprocal relationships between the amygaloid complex and most diencephalic nuclear masses render understandable the susceptibility of the amygdala to after-discharge arising from other sources.

In the experiments reported here, cortical zones of relative epileptogenesis were found to reside in or near most surface areas described as connecting with these diffusely projecting subcortical nuclei—particularly in frontal oculomotor fields, motor cortex, first temporal gyrus and tip, and cingulate gyrus. Diffusion of seizure activity from these zones, then, through rich subcortical as well as corticocortical connections, would be expected.

These experimental data, however, present certain exceptions to the above-described relationship between the epileptogenic tendency of a cortical locus and the nature of its subcortical connections. Anatomical and physiological connections are known to exist between the orbital, as well as the paraoccipital, regions of the cortex and the central cephalic brain stem [7]; yet these areas were found to exhibit relatively slight tendencies toward after-discharge or to disseminate seizure activity. It is possible, of course,

that the corticifugal projection from these regions is essentially inhibitory in action.[6] Contrastingly, the posterior parietal region displayed some epileptogenic activity, yet is not known to connect importantly with the diffusely projecting subcortical system studied here. It is evident, then, that direct corticosubcortical connections between excitable surface loci and the diffusely projecting brain stem nuclei investigated cannot account solely for the dissemination of all seizures induced by cortical stimulation. There must additionally be a capacity for the stimulated region to develop local after-discharge, a characteristic which appears to correlate well with other epileptogenic tendencies. Perhaps it is pertinent that neither the orbital surface nor the paraoccipital areas are known to have corticocortical connections with the highly epileptogenic motor cortex, while the parietal region referred to does have such connections.[35]

It is probable that other subcortical structures than those investigated in this study may contribute importantly to cortically induced seizure spread. We made relatively few recordings from the caudate nucleus and none from the putamen; yet the basal ganglia and other parts of the extrapyramidal system have been strongly implicated by Faeth and associates [20] and others § in the dissemination of after-discharge. It is clear from clinical observations and from experimental studies [16] that epileptic phenomena do not require the pyramidal tract for their development, their manifestation being effectively propagated by extrapyramidal pathways. In a recent study designed to investigate tracts to the basal ganglia, Jouvet and associates ‖ found that intimate connections existed not only between caudate nucleus, globus pallidus, and putamen and cortical zones strikingly similar to those diagrammed in Figure 5, but, further, that stimulation of these same cortical loci readily excited after-discharge in the basal ganglia. Presumably, then, the same cortical epileptogenic zones can effect diffusion of locally induced after-discharge through these channels involving the basal ganglia, as well as through the central cephalic brain stem.

There is as yet no obvious explanation for the relative differences in the threshold of excitability for spreading after-discharge from different cortical loci. It may be that the most active areas—parts of motor cortex, oculomotor fields, upper premotor region—discharge with greatest intensity because of intrinsic cortical mechanisms or because they interconnect most richly with other cortical and subcortical stations essential to the development of seizures. We are not prepared to distinguish between local cortical susceptibilities and special circuitry mechanisms but suggest that both may be important in the initiation and diffusion of epileptic discharge.

The demonstrated differences in susceptibility to cortically induced after-discharge have a number of additional implications. Susceptibility measured in this way may have some relationship to electroshock therapy: The most responsive structures, both cortical and subcortical, would appear to be the most vulnerable to currents delivered to the brain.¶ Even considering the peculiarities of current shunting through the head, it is evident that the differences in seizure thresholds of several times their value encountered in our experiments would make it highly probable that susceptible parts of the brain would be caused to initiate firing, and perhaps to undergo the greatest amount of discharge activity and subsequent fatigue. Perhaps uniform activation of the entire brain does not occur and is unnecessary for whatever beneficial effects are obtained by electroshock therapy. It is possible that some of the clinical differences in the effects of full-convulsive, abortive-convulsive, and non-convulsive shock therapy are related to the

§ References 15 and 16.

‖ Jouvet, M.; Ross-Duggan, J. K., and Terzuolo, C. A.: Cortifugal Connections to the Basal Ganglia in Cat and Monkey, in preparation.

¶ Walker, A. E.; Andy, O. J., and Poggio, S. F.: Role of Basal Ganglia in Convulsions, program abstract; 80th Annual Meeting, American Neurological Association, Chicago, June 13-15, 1955. Jung.[36]

degree to which certain cortical and subcortical structures are swept into activity.

Drug actions on the brain may be related to these obvious physiological differences in the discharge capacity of specific cortical and subcortical mechanisms. Certainly, medications found to induce alteration of seizure tendencies are known to have a primary effect on some of these structures; e. g., the barbiturates have been shown to depress selectively electrical activity within the reticular formation.[37] Moreover, distinctions in the treatment of grand mal, temporal lobe, and other types of epilepsy must concern the relative effectiveness of different drugs in suppressing abnormal activity in the particular cortical and subcortical structures implicated pathologically. Perhaps physiological assessment of selective drug action employing an experimental method similar to that utilized in this study might facilitate the development of agents which affect specific mechanisms disturbed in various clinical forms of epilepsy. If drugs are available or can be found which act upon these structures differentially, leaving the remainder of the brain essentially unaffected, perhaps another approach to the treatment of epilepsy may be available.

In conclusion, it seems evident that current concepts concerning epilepsy, at least experimental epilepsy, must be modified in order to accommodate two observations. 1. There appears to be an inherent capacity for the cells of certain cortical loci, and not others, to respond to excitatory or irritative stimuli by exhibiting persistent after-discharge. 2. Surface regions displaying this characteristic local response seem to have the capacity secondarily to excite certain diffusely projecting subcortical structures which are capable of disseminating the induced discharge widely. By such means, perhaps, epileptogenic loci can interrupt normal cerebral function. It may be supposed, therefore, that any irritative lesion in the cortex will tend to give rise to one or another of the manifestations of epilepsy, the closer it is situated to any of these inherently epileptogenic areas.

SUMMARY

Experiments were performed on eight monkeys in order to investigate the capacity of different cortical regions to initiate spreading seizure discharge upon direct stimulation.

It was found that the "epileptogenic" cortical zones were located in portions of the motor and premotor cortex, posterior cingulate region, superior temporal gyrus and tip, and parietal area. Other surface areas displayed little or no propensity to develop spreading after-discharge.

In these experiments, the subcortical nuclei most commonly involved in the cortically induced seizures were the reticular formation, septal region, and amygdala.

An inherent tendency of cells in the described cortical loci to respond to local stimulation by exhibiting after-discharge, and their functional relations with subcortical nuclei known to exert diffuse influences upon the central nervous system, may both be important factors in the initiation and spread of seizures.

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A Psychiatric Study of Attempted Suicide in Persons Over Sixty Years of Age

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During the course of a study of attempted suicide, it became evident to us that there were many differences between younger and older patients who have attempted suicide. The work reported here was undertaken to investigate in greater detail the differences between these two groups and to study the characteristics of suicide attempts in persons in the older age group.

Special features of suicide attempts in the older persons will be described in this report. These features are important in the diagnosis and practical management of these patients, especially in preventing a subsequently successful suicide. Additional evidence that there are special features in the suicide attempts of older people will be supplied by comparing data from this study with a similar one from the United Kingdom.[1]

METHOD OF STUDY

The present report is part of a larger study of attempted suicide.[2] In the larger study, 109 patients who had attempted suicide were examined. There were 19 patients aged 60 and over. In this report these 19 older patients are described in detail. In addition, this older group is compared with the younger group of the remaining 90 patients aged 59 and under. The method of selecting the patients, the method of obtaining data about the patients, the diagnostic criteria, and the authors' evaluation of the seriousness of the suicide attempt have all been described in detail elsewhere.[2] All patients included in the total study were examined by means of an open-ended questionnaire, which covered past and present medical history, personal and social history, and details about the suicide attempt. In addition, part of the questionnaire was designed to elicit the history and symptoms, if present, of the psychoses, psychoneuroses, and sociopathic personality disturbances (psychopathic personality; chronic alcoholism). The seriousness of the attempt was ascertained by an assessment of the actual medical or surgical condition of the patient resulting from the attempt, as well as by a clinical evaluation of the seriousness of the patient's intentions by the psychiatrist. Statistical methods used have been described.[3] A significance ratio of 2.0 or greater, corresponding to odds of about 20 to 1, was accepted as being statistically significant.

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RESULTS

General Characteristics.—In the total group of 109 patients, there were 19 patients aged 60 and over. The age range in this older group was from 61 to 84 (Table 1). The average age of the older group was 71, that of the men was 73, and of the women 66. Fourteen of the patients were men and five were women (Table 1). These patients had completed an average of six years of school.

The Suicide Attempt.—All patients in the older group except one (Patient 5, whose memory was so poor she had forgotten the attempt) readily admitted their suicide attempts. The methods of attempting suicide in the older group of 19 patients (Table 1) were generally violent ones (cutting, leaping, shooting, drowning, and choking). This was true for 79% of the men and 60% of the women.

Twelve of the older patients (63%) had made serious attempts. This high proportion

TABLE 1.—*Clinical Description of the Nineteen Patients* *

Patient No.	Sex	Age, Yr.	Method of Attempting Suicide	Serious (S) Versus Not-Serious (NS) Suicide Attempt	Symptoms of Present Illness †	Duration of Present Illness, Mo.	Presence of Serious Medical or Surgical Illness	Previous Psychiatric Illnesses	Length of Follow-Up, Mo.	Condition at Follow-Up
Group I—Chronic Brain Syndrome (Dementia)										
1	M	79	Cutting	S	1, 2, 3, 7, 26, 27, 28, 29, 30, 36, 37, 39	36	No	Alcoholism (?)	24	Hospitalized (mental)
2	M	75	Cutting	S	2, 14, 15, 26, 28, 29, 37, 38	24	Yes	None	2	Dead
3	M	72	Leaping	NS	4, 20, 29, 30, 41	60	Yes	None	6	Dead
4	M	71	Drugs	S	1, 4, 9, 16, 18, 25, 36	72	Yes	None	1	Dead
5	F	69	Leaping	NS	18, 19, 26, 36	16	Yes	None ‡	15	Hospitalized (mental)
Group II—Manic-Depressive Psychosis, Depressed Phase										
6	M	78	Cutting	S	5, 7, 20, 26, 27, 28, 29, 30, 31, 32, 33, 36, 37	6	Yes	None	18	Hospitalized (general)
7	M	73	Shooting	S	10, 14, 15, 20, 26, 28, 29, 30, 34, 36, 37	48	No	None	29	Sustained depression
8	M	64	Cutting	NS	10, 13, 14, 15, 20, 21, 23, 26, 28, 29, 30, 33, 35, 36, 37, 41	9	No	M-D D(2) §	18	Working
9	M	65	Gas	NS	3, 7, 8, 9, 10, 15, 18, 23, 26, 27, 28, 29, 30, 31, 33, 35, 37, 39, 43	2	No	M-D D(1)	30	Retired but well
10	M	71	Cutting	S	‖	1	No	M-D D(1)	¼	Dead (suicide)
11	F	65	Drugs	S	1, 2, 3, 4, 5, 6, 10, 11, 15, 17, 18, 19, 21, 23, 24, 25, 26, 27, 31, 33, 35, 36, 37, 39, 40	18	No	None ‡	24	Recurrent depression
12	F	69	Drugs	S	2, 4, 7, 8, 9, 10, 14, 15, **23, 24, 26, 28,** 30, 31, 33, 34, 37, 39, 41	12	Yes	M-D D(2)	6	Dead

13.	F	64	Drowning	S	5, 11, 12, 14, 15, 16, 23, 26, 27, 28, 29, 30, 31, 32, 33, 35, 36, 37, 38, 41	18	No	M-D D(1); alcoholism	17	Manic attack; then working
14.	F	61	Choking	S	10, 15, 26, 27, 28, 29, 30, 31, 32, 33, 34, 36, 39, 41, 42	2	No	M-D D(1)	17	Working
				Group III—Acute Brain Syndrome (Toxic Psychosis)						
15.	M	77	Leaping	NS	8, 13, 14, 15, 18, 21, 22, 26, 27	1	Yes	Alcoholism	18	Working
16.	M	73	Cutting	NS	15, 31, 36	3	Yes	Alcoholism	24	Retired but mentally well
17.	M	61	Drugs	S	5, 10, 18, 26, 28, 29, 30, 33, 39	3	Yes	Alcoholism	2	Dead (suicide)
				Group IV—Chronic Alcoholism						
18.	M	84	Cutting	NS	4, 14, 26, 27, 28, 29	120+	No	None	27	Retired
19.	M	73	Cutting	S	29	600+	No	None	34	Working

* The mental status findings for the 4 groups may be summarized as follows: Group I—Every patient was disoriented for time and one of them for place also. Four of them were hallucinated and two deluded. Memory was markedly impaired in all. Every patient had evidence of intellectual deterioration. Group II—All of these patients were depressed. All showed motor retardation, slow and low-voiced speech, crying, and/or agitation. None of these patients showed any disorientation, memory loss, or intellectual impairment. There was one hallucinated patient. Four showed typical depressive delusions (text). Group III—The three patients were hallucinated, deluded, and disoriented for time and place. The delusions were poorly organized and changing. All had a serious medical or surgical illness with fever. Group IV—These patients had no signs of psychosis. Both had a prolonged history of excessive intake of alcohol.

† The numbers in this column refer to the following symptoms:

1. Headache
2. Blurred vision
3. Diplopia
4. Dizziness
5. Lump in throat
6. Aphonia
7. Dyspnea
8. Palpitation
9. Chest pain
10. Anorexia
11. Nausea
12. Vomiting
13. Abdominal pain
14. Constipation
15. Weight loss
16. Urinary frequency
17. Menstrual pain
18. Sexual indifference
19. Sexual frigidity
20. Impotence
21. Paresthesias
22. Fainting spells
23. Pain in back
24. Pain in joints
25. Pain in limbs
26. Nervousness
27. Cry easily
28. Easily upset
29. Depressed episodes
30. Apathetic
31. Lack of energy
32. Hyperactive
33. Tires easily
34. Diurnal variation of depression
35. Crowd phobia
36. Chronic unhappiness
37. Insomnia
38. Nervous chills
39. Weakness
40. Difficulty walking
41. History of changed behavior
42. Phobias

‡ These two patients described themselves as having been nervous most of their lives, but this was not a diagnosable condition.
§ M-D D indicates Manic-depressive psychosis, depressed phase. Figures in parentheses indicate number of previous attacks.
‖ This patient committed suicide before the questionnaire was completed.

of serious (genuine) attempts is also reported in other studies of attempted suicide in old persons.[1]

Eight of these patients (42%) stated they had had suicidal thoughts, varying in duration from one year to a few hours. However, only three (16%) had actually told anyone of these suicidal thoughts. At the time of their attempt, 13 of these patients were under a physician's care. While eight patients had told their doctors about their depressed feelings, none had told his physician about his suicidal ideas. It is our impression, as we have previously stated,[2] that the patient's reticence to talk about suicide results mainly from the physician's failing to ask him directly about the possibility. Most patients, when asked about suicidal ideas, will readily discuss them.[4]

The immediate precipitating factors in the suicide attempts were investigated by questioning the patients about the "main reason" why they attempted suicide. As might be expected, feelings of depression and melancholy were the most frequent reasons given. It was remarkable, however, that 11 of the 19 patients failed to relate their feelings of depression to social, personal, or medical problems; 6 of these patients gave answers indicative of depressed fee'ings, such as, "I'm tired of living; I can't collect my thoughts," or "I felt bad, was nervous and blue," or "I've had a fit of depression." Another four patients gave depressive delusions, such as "I've got a pain in my mouth, and nobody can help me" (somatic delusion), or "I'm destitute and ruined" (delusion of poverty in a well-to-do man). One patient did not remember the attempt (amnesia associated with a senile psychosis). The remaining eight patients did mention personal, social, or medical problems as their chief reasons for attempting suicide; three gave the death of a loved one, three gave financial worries, one gave the pain and disability associated with a recent hip fracture, and one gave concern over his alcohol addiction.

Although most (58%) of the patients did not relate their suicide attempts to specific problems in their lives, we considered the possibility that the recent occurrence of such problems might have contributed to their suicide attempts, even though the patients did not themselves make the connection between the problems and the suicide attempts. Therefore, each of the 19 patients was asked about potentially disturbing factors in his life during the six months preceding his suicide attempt (Table 2). Sixteen such factors were investigated (Questions 14-29 in the

TABLE 2.—*Comparison of the Incidence of Possibly Disturbing Events and Symptoms During the Six Months Prior to the Suicide Attempt in the Older and Younger Groups*

	Older Group (19)	Younger Group (90)	Significance Ratio
Social (interpersonal) stress			
Death of relative or friend	16*	17	<2
Change of residence	11	38	3.0
Friction with parent, sibling, or child	5†	17	<2
Friction with spouse or lover	0	67	13.5
Impending break-up of marriage or love affair	0	26	5.6
Divorce, separation, or desertion	0	17	4.3
Other sexual difficulties	0	10	3.2
Legal difficulties	0	8	2.5
Job and financial difficulties			
Job difficulties	16	26	<2
Financial problems	16	12	<2
Medical problems			
Serious medical or surgical illness	53	4	4.2
Mental state and/or symptoms			
Delusions	47	13	2.8
Drinking just before suicide attempt	16	44	2.8
Feeling of having been disgraced	5	18	2.9

* Six patients (total, 32%) mentioned the death of a relative within the past five years.
† Three patients (total, 16%) mentioned such friction within the past two years.

previous paper in this series [2]). It is noteworthy that none of the social or financial problems occurred in a high proportion of the older patients within six months of the suicide attempt. Medical problems occurred in over half the patients. The occurrence of delusions in 47% of the older patients is related to the high percentage of psychotics in this group, as will be apparent in the next section.

Diagnostic Features of the Group.—It is often emphasized that suicide attempts in elderly people are primarily reactions to age, loneliness, and physical illness, while the importance of a diagnosable psychiatric disease is overlooked. In this study, every patient was found to be suffering from a definite psychiatric disease. These patients had diseases which fell into four diagnostic categories (Table 1): (1) chronic brain syndrome (five patients); (2) manic-depressive depression (nine patients); (3) acute confusional state (three patients); and (4) chronic alcoholism (two patients). These diagnoses were based on the symptoms of the present illness (Table 1), history of past episodes of mental illness (Table 1), findings on physical examination, mental status findings (Table 1), laboratory studies, and a follow-up investigation.* Psychiatric diagnoses are often considered to be vague and subject to the individual examiner's bias. However, we believe that the data reported in Table 1 (Group I—typical mental status findings of organic brain disease without recovery; Group II—typical symptoms, present illness, and mental status findings of psychotic depression; Group III—typical mental status findings of organic brain disease with recovery Group IV—chronic excessive alcoholic intake without other psychiatric illness) and the checking of the diagnosis by follow-up study insure that these diagnoses are probably correct.

There are three noteworthy points about the diagnoses of these older patients: (*a*) There were no patients without diagnoses; (*b*) 89% of these patients were psychotic, and (*c*) of the 15 or so major diagnostic categories in psychiatry (for example, schizophrenia, anxiety neurosis, manic-depressive psychosis, etc.), only 4 categories accounted for the diagnoses in all of these 19 patients.

Previous Psychiatric Illness (Table 1).—Of the nine patients with a manic-depressive depression, six had had a previous episode of depression from which they had recovered. Two patients had never been psychiatrically ill prior to the present episode, and one patient had had symptoms suggestive of but not diagnostic of conversion reaction (hysteria). Of the five patients with a chronic brain syndrome, three had never been psychiatrically ill prior to the onset of the syndrome, but one stated that she had been "nervous." One of these patients may have been an alcoholic in his youth but had not drunk for at least 20 years prior to his suicide attempt. Of the three patients with an acute brain syndrome, all had a prior history of chronic alcoholism, although the examiners did not believe the acute brain syndrome preceding the suicide attempt was directly related to the chronic alcoholism. One of these patients was febrile and had pulmonary tuberculosis, another had severe generalized arteriosclerosis necessitating a leg amputation sometime later, and the third was not taking alcohol excessively at the time he developed his acute brain syndrome.

Physical Illness.—Nine of these patients (Patients 2, 3, 4, 5, 6, 12, 15, 16, and 17) had definite physical illnesses in addition to their psychiatric illness. Two patients (12 and 15) had hypertension and cardiac failure; one (Patient 2) had tabes dorsalis and generalized arteriosclerosis; and another (Patient 3) had had a stroke two to three years previously, which had left some residual paresis. Patient 4 had generalized arteriosclerosis, which was severe enough

* It became apparent at follow-up that three patients who were originally diagnosed [2] as suffering from a chronic brain syndrome had actually had other illnesses: an acute brain syndrome, chronic alcoholism (having presented with an acute demented state related to excessive alcoholic consumption), and a manic-depressive depression, respectively.

that one leg became gangrenous and amputation was necessary. Patient 6 had suffered from severely disabling arthritis for four or five years. Patient 16 had generalized arteriosclerosis and had fractured his right hip four years previously. The result of the hip reduction was not satisfactory, and he was almost an invalid. The remaining patient with an illness was Patient 17, who three months previously had consulted a physician because of fatigue and weakness and had been discovered to have pulmonary tuberculosis. It is interesting to note, however, that despite the severe and disabling character of these illnesses, only three of these patients in any way related their attempts to their illnesses, and only one of them gave his physical illness as a chief reason. It would seem, therefore, that even though physical illness occurs frequently in these people, their reaction to it is not an important immediate factor in their suicide attempts.

Personal and Social History.—In general, although there were too few patients to be certain, there was nothing exceptional about the personal histories of these people. One-quarter (five patients) of these older patients had experienced a broken home during their childhood, before age 15. In all but one case, separation from the parent or parents was by death rather than divorce.

The marital history of these older patients was probably not vastly different from that of the population at large. Only one had failed to marry. Two of the patients had separated from their spouses—one after 25 years of marriage and another after 10 years of marriage. Three (16%) had divorced their spouses after fairly long marriages. One of these had married for a second time (happily).

The majority of the older patients had good job histories in that they had worked for many years at the same jobs and had worked steadily. Only three (Patients 4, 6, and 15) had at some period in their lives a variety of jobs, which they left for no adequate reason, but even these three had each held one job for at least 12 years. The occupational level was known for 18 of the 19 patients. Nine of these patients were unskilled workers, two were farmers, two were skilled workers, four were "white collar" workers, and one was a businessman. Thus, 61% of the patients were below the level of skilled workers, and 39% were skilled workers or higher.

Only three patients gave loneliness as a reason for their suicide attempts. In each case, the loneliness had resulted from the death of a loved one. Additional evidence that loneliness and social isolation may not have been important factors leading to the suicide attempts of the majority of these patients is indicated by the fact that 12 of the 19 patients were living with either their spouses (6 patients), their children (4 patients), or their siblings (2 patients). There was little evidence of excessive friction in any of these family situations. One patient was hospitalized, two were living in homes for the aged, and four were living alone.

Comparison of Older and Younger Groups (Table 3).—The sex distribution of the older group (74% men) differed from that of the younger group (39% men). The significance of this finding is uncertain, since in other studies of consecutive cases of attempted suicide, some have reported a preponderance of men in the older group,† while others have reported a preponderance of women.‡

There were no undiagnosed patients in the older age group. In contrast, it was not possible to make a definite diagnosis in 23% of the patients in the younger group. For the older age group, the patients were in only 4 diagnostic groups, whereas in the younger age group they were in 10 diagnostic groups. In the older age group, 89% of the patients were psychotic, whereas in the younger group only 23% of the patients were psychotic. Apparently, then, in the younger age group, there is a wide variety of psychiatric reaction types, whereas in older groups the diagnoses are definite and limited to a few diagnostic categories (chiefly psychotic).

† References 5-7.

‡ References 1 and 8.

The suicide attempts of the older group were more often serious than those of the younger groups (63% versus 26%). The rarity of complaints about loneliness, finances, or hopeless physical conditions in this older group makes us doubt that their greater incidence of serious attempts is simply a reflection of the greater difficulties that older people suffer, either as a result of social isolation or poor health. Rather, as we have indicated in a previous paper,[2] the higher frequency of serious attempts in the older group is related to two facts: Serious suicide attempts occur more frequently in patients with manic-depressive psychosis and chronic brain syndrome than in other psychiatric illnesses, and these two diseases occur more frequently in older people.

When the older group was compared with the younger group with regard to possibly disturbing antecedent factors (Table 2), the following striking difference was noted: Potentially disturbing life situations were much more frequent in the younger group than in the older group. The only disturbing situations which occurred more frequently in the older group were the presence of a serious medical or surgical illness and the occurrence of delusions. Problems related to love and marriage were very frequent in the younger age group. The complete absence of any serious current marital problems in the older group is striking, although a low incidence might be expected.

Result of Follow-up (Table 1).—Six patients had died within six months of the time they had originally been examined: two by suicide (Patients 10 and 17), and one each from tabes dorsalis (Patient 2), generalized arteriosclerosis (Patient 3), arteriosclerotic gangrene of the leg (Patient 4), and arteriosclerotic heart disease with congestive failure (Patient 12). A detailed description of the two patients who subsequently committed suicide has been presented elsewhere.[2]

TABLE 3.—*Comparison of the Older Group with the Younger Group*

	Older Patients	Younger Patients	Significance Ratio
Number of patients	19	90	...
Mean age	71	33	...
Age range	61-84	12-59	...
Sex distribution			
Men	74%	39%	3.1
Women	26%	61%	3.1
Diagnosis			
Definitely diagnosed	100%	77%	5.2
Not diagnosable	0%	23%	5.2
Proportion with psychosis	89%	23%	7.8
Number of different diagnostic groups	4	10	...
Method of attempting suicide			
Violent means			
Men	79%	54%	2.4
Women	60%	24%	3.0
Seriousness of suicide attempt			
Serious	63%	27%	3.1
Not serious	37%	73%	3.1
Evidence of previous maladjustment			
Mean number of mental hospitalizations	0.32	0.97	2.5
Mean number of general hospitalizations	1.53	3.40	3.0
Proportion divorced at least once	16%	37%	2.1
Proportion from broken parental homes (before age 15)	26%	62%	3.2

The mean length of follow-up for the 13 surviving patients was 23 months. At the time of follow-up, five of these patients (38%) were working and another two (15%), although retired, were mentally well. Thus, over half (53%) of the living patients were working or, if retired, were mentally well and able to live at home. This underlines the importance of preventing or treating ade-

quately these suicide attempts and the diseases which lead to them, for even in this age group a significant number of patients again became useful citizens.

COMMENT

The most important finding of this study, we believe, is that every elderly patient who attempted suicide had a clinically diagnosable (evident) psychiatric illness prior to his attempt. A second important finding related to diagnosis was that 89% of these patients had a psychotic illness—chronic brain syndrome, acute brain syndrome, or psychotic depression (Tables 1 and 4). A third finding was that two illnesses—psychotic depression and chronic brain syndrome—accounted for almost three-quarters (73%) of the patients.

A very high proportion (63%) of these elderly patients made serious suicidal attempts, as judged by their medical or surgical condition and by the psychiatrists' evaluation of the seriousness of their intention. That these judgments were probably valid is indicated by the fact that two of these patients killed themselves within the year following their attempts.

There are two important points in the proper management of these patients by their own physician which become clear from a consideration of the above points. First, all these patients have a clinically evident illness and, therefore, the physician without special training in psychiatry will be able to diagnose them. This is particularly true because, in contrast to many other psychiatric illnesses, the psychiatric diseases occurring in this group of older patients are clearly defined clinical syndromes. Secondly, once the disease is recognized, an already defined management procedure is at hand. The majority of these patients should be hospitalized in order to prevent a successful suicide and in order to treat the underlying illness. Treatment of the underlying illness is of especial importance, since two of these syndromes—psychotic depression and acute brain syndrome—are treatable and reversible.

A recent study of Batchelor and Napier,[1] in Edinburgh, Scotland, done independently and almost simultaneously with our study,[2] adds strong support to these conclusions. The Edinburgh study is exactly comparable to the present one: In both instances (*a*) consecutive patients who were brought to a large general hospital after having attempted suicide were studied; (*b*) they were systematically interviewed and examined immediately after their attempts; and (*c*) the original examiners obtained follow-up information about these patients at least one year after the initial examination.

These studies are compared in Table 4. First, in regard to diagnosis, every patient in both series had a condition in which a definite psychiatric diagnosis could be made. This contrasts with the difficulty in making a definite diagnosis in younger patients. Batchelor and Napier state, "Cases of attempted suicide in this [older] group are more easily classified than those occurring in people in the first half of life. The persons here are usually definitely mentally ill. . . ." § The patients not only have a definite psychiatric illness, but the vast majority are psychotically ill (82% in Batchelor and Napier's series, 89% in the present one). Batchelor and Napier state, ". . . at least two-thirds are certifiable as insane, and the personalities of the majority are clearly involved to a psychotic extent." It is striking that patients with one of two syndromes, chronic brain syndrome or psychotic depressive reaction, constitute approximately three-quarters (80% and 73%, respectively) of the older patients who attempt suicide. (It should be pointed out here that chronic brain syndrome signifies senile psychosis or cerebral arteriosclerosis, as there was only one patient in either series who had a chronic brain syndrome associated with another illness [taboparesis].)

§ In a personal communication, Dr. Batchelor has amplified this by stating, "I thought that all the old patients that I saw had a clinically diagnosable condition at the time of the attempted suicide."

The striking agreement between these two studies, done independently and in different countries, increases the evidence that a clinically diagnosable illness almost always precedes an attempted suicide in elderly patients. This suggests that factors often thought to be important in depressive states and attempted suicide in older persons, such as loneliness, "reactions to the aging process," feelings of uselessness, enforced unemployment, and presence of serious physical illnesses, are probably not alone sufficient to impel an older person to make a suicide attempt. A definite psychiatric illness almost always intervenes. This opinion has been expressed by Roth and Morrissey,[10] who state, ". . . it is not . . . argued that social factors play no part, but that it is through their role in the precipitation of depressive psychoses that they contribute to the causation of suicide in the old." However, in the present study, 8 of our 19 patients had illnesses (acute and chronic brain syndrome) which are presumably not precipitated by social factors, and 4 of them had an illness which began well before the age of 60—2 chronic alcoholics and 2 of the manic-depressives. Thus, almost two-thirds (12 out of 19) had illnesses which could not have been precipitated by social factors peculiar to old age. This is not to deny, however, that the suicide attempt itself may not sometimes be precipitated by social factors impinging on a patient already mentally ill.

Other comparisons with the Edinburgh study (Table 4) also show many areas of close agreement. Batchelor and Napier also believe, as we do, that most suicide attempts in old age are serious, that is, genuine. They state, "There is rarely any doubt that the suicidal attempt of an old person has been a genuine one." Whether the conclusions from this study and the Edinburgh study are valid for the business and professional group is not known, since in these studies there was a total of only four patients in this class.

TABLE 4.—*Comparison of the Present Study with That of Batchelor and Napier* [1]

	Batchelor and Napier	Present Study
Number of patients	40	19
Diagnosis		
Patients in whom a definite diagnosis could be made	100%	100%
Diagnostic groups:		
Depression	70%	47%
Chronic brain syndrome	10	26
Acute brain syndrome	0	16
Chronic alcoholism	10 *	11
Psychopathy (sociopathic personality)	8	0
Paranoid state	2	0
Occupational status		
Skilled worker and higher	55%	39%
Below skilled worker	45	61
Previous mental health		
History of diagnosable mental illness	55%	53%
Questionable history of mental illness	20	21
No history of previous mental illness	25	26
Antecedent difficulties		
Presence of serious chronic physical illness	45%	47%
Living alone or in hostel or old-age home †	22	37
Change in social status or living condition	10	11
Death of a loved one	27	16
Financial problems	15	16
Suicide attempt because of hatred or spite	7	5
Prior and subsequent suicidal acts		
Previous attempts	17%	11%
Subsequent attempts (1-year follow-up)	3	11
Subsequent successful suicide (1-year follow-up)	5	11

* This figure was computed from the data of Batchelor,[9] using our criterion that chronic alcoholism with a nonpsychotic depression was classified as alcoholism.
† 78% of Batchelor and Napier's and 63% of our patients were living with their families.

The antecedent factors noted in Table 2 and discussed in the text indicate that the factors leading to suicide in the older group are not purely social and frequently do not depend on individual reactions to the social environment. In contrast suicide attempts in the younger group were most often precipitated by factors of a social or personal nature. It is our impression that the difference between the younger and older groups is not related merely to age, but reflects a more fundamental difference, that in the larger areas of social functioning the older people throughout their lives have performed significantly better than the younger group. This idea is supported by the facts (Table 3) that, although the older group averaged 38 years older, they had fewer mental hospitalizations, fewer general hospitalizations, and fewer divorces. This apparently better social adjustment may be associated with the fact that as children the older group experienced broken homes much less frequently than the younger group (Table 3).

This has been a study of patients who attempt suicide and not a study of patients who commit suicide. We do not know, therefore, how many of these findings and conclusions apply to a group of patients who actually commit suicide. We tend to believe that the patients whose attempts we designated as "serious" would be very similar clinically to patients who commit suicide.

SUMMARY

Nineteen patients aged 60 and over were interviewed and examined immediately following a suicide attempt. These 19 patients were part of a larger study of 109 patients who had attempted suicide.

Every one of the 19 older patients had a clinically diagnosable illness. There were nine patients with a psychotic depression, five with a chronic brain syndrome (dementia), three with an acute brain syndrome (toxic psychosis), and two with chronic alcoholism. Thus, 89% of these patients had a psychotic illness. These findings concerning diagnosis contrasted with those in the younger group, aged 59 and under.

The majority of the suicide attempts in the elderly patients were serious (genuine).

Most of these patients suffered from a treatable (psychotic depression) or reversible (acute brain syndrome) disease. Therefore, many of them can be returned to society as useful citizens if a subsequently successful suicide is prevented.

The immediate management of these patients consists of hospitalization in order to treat the underlying illness and to prevent a successful suicide.

A follow-up study of 23 months showed that two patients, one of whom had a psychotic depression and the other an acute brain syndrome, had committed suicide.

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Palatal Myoclonus

Further Clinical and Pathophysiological Observations

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The clinical characteristics and pathophysiology of the phenomenon of palatal myoclonus are not yet clearly understood. Since 1886 case reports of this phenomenon have appeared in the literature periodically,* and, although the rate and other physical characteristics have been described, little can be found concerning observations of these movements under various conditions, both spontaneous and induced, that might distinguish them from other abnormal motor phenomena. Also, there has been a relative paucity of satisfactory postmortem studies. Evidence based on approximately eight adequate pathologic reports points to the inferior olive as the structure primarily implicated in the production of these abnormal movements. Except for Freeman's case,[7] in which a lesion in the central tegmental fasciculus was found, the major pathology was located in one or both inferior olives. One inferior olive usually appeared "swollen" as compared with the other, and, therefore, the term "pseudohypertrophy" was used. Although other structures, such as the dentate and red nuclei, have been mentioned as being involved in the production of palatal myoclonus,[8] their full significance has not been demonstrated.

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The material was collected from the neurologic services of the Mount Sinai Hospital, and Bellevue and Goldwater Memorial Hospitals (New York University).

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* References 1-6.

On the other hand, evidence is submitted here that the inferior olive is not the primary site necessary in producing these abnormal movements; rather, that the major lesion may be in one cerebellar hemisphere (particularly in the region of the dentate nucleus) and only secondarily involve the opposite inferior olive. In addition, further clinical observations have been made on patients with myoclonic movements of the palate and related structures that point up its unique behavior, as compared with other abnormal involuntary movements. The present study is an outgrowth of the investigation reported in 1952 by Bender, Nathanson, and Gordon,[13] touching on some of these points.

MATERIAL AND METHOD

Twelve patients with constant abnormal involuntary movements of the palate, pharynx, tongue, and lips were studied. Four of these patients died, and postmortem examinations were performed on three. One of these cases already had been reported, as has Case 6.[13] In one other patient of this group presumptive anatomic localization was obtained at operation.

Clinically, observations were made under the following conditions: (1) natural sleep; (2) during phonation and voluntary breath holding; (3) following administration of intravenous barbiturates prior to and during induced sleep; (4) caloric stimulation of the ears; (5) carotid sinus pressure; (6) in two cases, during coma and impending death, and (7) in another, during inhalations of 100% O_2 and 5% CO_2.

In several instances electrical recordings of these abnormal movements were made simultaneously with the heart beat, abnormal eye movements, or other types of abnormal involuntary movements, when they existed. The effect of intravenous barbiturates also was recorded by this method.

REPORT OF CASES

Case 1.—A man aged 22 was admitted to the Mount Sinai Hospital on Nov. 6, 1948, with a two-week history of sudden onset of headache, nausea, and vomiting. One week later he complained of

transient diplopia, and on the following morning there was weakness of the right side of the face. On admission he showed paralysis of the right side of the face of peripheral type, weakness of conjugate ocular gaze to the right, ataxia of the right extremities, and decreased auditory acuity on the right. Caloric tests with cold water revealed a nonfunctioning labyrinth on the right. While the patient was under observation, there developed weakness of the right lateral rectus muscle and diplopia on central gaze. Within three weeks there was improvement. The paresis of right conjugate gaze, the weakness of the right lateral rectus, and the right facial paresis disappeared almost completely, while the ataxia of the right extremities lessened. The cerebrospinal fluid showed 8 lymphocytes per cubic millimeter and a total protein of 88 mg. per 100 cc. The impression was that the patient had acute encephalitis, but the possibility of multiple sclerosis was considered.

Several days after his discharge from the Mount Sinai Hospital there was a sudden onset of weakness of the left side of the face, which disappeared within one week. During the following six months the patient was relatively asymptomatic except for an occasional episode of double vision and the persistent, but slight ataxia of the right upper and lower extremities.

In the middle of September, 1949, he began to complain of numbness and weakness of both feet, and within the next five days there developed complete paralysis of the lower extremities, with urinary and fecal incontinence. He was admitted to Bellevue Hospital on Sept. 21, 1949.

The past history revealed that from the ages of 9 to 12 years the patient's left lower extremity was in a cast because of suspected "tuberculosis" of the left knee joint. Since the age of 14 he had had a nonproductive cough. In 1945 he was rejected by the draft board because of a "suspicious chest x-ray." During the next two years repeated examinations of the sputum and x-ray examinations of the chest showed no evidence of tuberculosis. He was told he had "unresolved pneumonia."

The general physical examination on his admission to Bellevue Hospital was normal except for an indurated area, approximately 3 to 5 cm., just to the right of the midthoracic portion of the spine.

The neurologic examination disclosed a coarse, rapid nystagmus in all directions of gaze, including central fixation. The nystagmus on direct forward gaze was in the vertical plane. The patient complained of objects bobbing "up and down" on central fixation at a far point (oscillopsia). Slight ataxia of the right upper extremity was noted. There were flaccid paraplegia, absence of the left knee jerk, and depression of the other tendon reflexes. A well-demarcated sensory defect for all modalities was evident below the level of the seventh thoracic spinal segment.

There were rhythmic, continuous movements of the right side of the palate and pharynx, at a rate of 130 per minute. Slight movement of the right side of the pharynx also was noted. The right posterior pharyngeal wall moved to and fro, and the right side of the palate and the right faucial pillar moved synchronously from side to side. Phonation did not affect the movements. The larynx and diaphragm were not involved. The palatopharyngeal movements showed no synchrony with the nystagmus on direct forward gaze or with the pulse rate. The palatal movements persisted during sleep. The patient denied awareness of the movements, and the examiner could not detect audible "clicking" at close range. The right palatopharyngeal myoclonic movements persisted without change in character until death.

Effect of Amobarbital Sodium on Rhythmic Movements of the Palate and Eyeballs.—Intravenous injection of amobarbital sodium failed to alter the rate and amplitude of the palatal and pharyngeal movements. On the other hand, it abolished the oscillopsia and nystagmus on forward gaze and increased the nystagmus on lateral and vertical gaze. Approximately 5 grains (0.324 gm.) of amobarbital sodium was injected intravenously within four minutes. Three minutes after the injection was started, the nystagmus on direct forward gaze and the oscillopsia disappeared. Five minutes later there was no response to stimulation with the opticomotor drum, whereas prior to the drug a typical ocular response was elicited. Twenty minutes after the injection was started the nystagmus on direct forward gaze returned, but it was not accompanied by the return of the oscillopsia until approximately 15 minutes later. During the entire observation period there was shimmering nystagmus on horizontal gaze to either side. There was no alteration of the palatal myoclonus.

Caloric Stimulation.—Stimulation of each auditory canal with cold water failed to produce visible change in the character and rate of the ocular nystagmus. There was no vertigo or nausea. The palatopharyngeal myoclonus was not affected by the caloric stimulations.

Laboratory Studies and Operation.—Spinal puncture shortly after admission disclosed xanthochromic spinal fluid, containing 200 red blood cells per cubic millimeter. The total protein was 864 mg. per 100 cc. Only a small amount of fluid could be obtained, and adequate manometric tests were impossible. A cisternal myelogram showed a complete block of the iophendylate (Pantopaque) at the level of the sixth thoracic vertebra. The cisternal fluid was clear and colorless, and the total protein was 39 mg. per 100 cc. Roentgeno-

grams of the chest taken in the supine position showed obliteration of the left lateral costophrenic sulcus by "thickened pleura or minimal effusion." The thoracic, lumbar, and sacral roentgenograms appeared normal except for "suggestive inflammatory changes" involving the left eighth rib at its vertebral extremity. All other pertinent laboratory findings were normal.

The subcutaneous mass to the left of the spine in the midthoracic region was noted to be larger and more indurated than it has been on admission. A biopsy diagnosis of this tissue was reported as reticulum-cell sarcoma. A laminectomy was performed, and similar tumor tissue was subsequently located in the extradural and intradural regions at the level of the fifth to the eighth thoracic vertebrae. On opening the dura, it was noted that the spinal cord was congested, discolored, and swollen. After the laminectomy the course was downhill, and the patient died on Jan. 16, 1950. Several minutes prior to his death, when the patient was semistuporous, it was noted that the palatopharyngeal myoclonus was present and exhibiting the same rate and character as on previous examinations.

Autopsy Findings.—The brain weighed 1260 gm. The dura showed an irregular, grayish, elevated, indurated area, approximately 2 by 3 cm., on the inner surface of the right parieto-occipital region. Cut sections showed that tumor tissue infiltrated the entire thickness of the dura. The sagittal sinus was patent. The cerebral hemispheres were symmetrical. There were no areas of softening. The arachnoid membranes were slightly opaque. No abnormalities of the gyri, sulci, or vessels on the surface of the brain were noted. On section of the cerebrum, cerebellum, and brain stem no gross abnormalities were detected.

The dura of the spinal cord was thick and adherent to the midthoracic region. It presented indurated, light-grayish, raised, irregular areas. Section through this area showed sarcomatous tumor tissue infiltrating the entire dura, the arachnoid, and the posterior aspect of the cord.

Tumor tissue was also noted in the lungs, kidneys, testes, and liver. These organs presented irregular, large and small, grayish, indurated areas.

Microscopic examination of serial sections of the entire brain stem disclosed conspicuous changes in the left inferior olivary nucleus, the right dentate nucleus, and the right restiform body (inferior cerebellar peduncle). These can be seen with the naked eye in Figure 1. In addition, the left inferior olivary nucleus appeared larger than the right.

Nissl, cresyl violet, and hematoxylin-eosin stains showed a decrease in the number of ganglion cells within the left inferior olivary nucleus; rarefaction, congestion, and small hemorrhages in the region of the right dentate nucleus; gliosis in this region, and a distinct decrease in the number of Purkinje cells in the right half of the cerebellum. There was round-cell infiltration, particularly in the region of the right dentate nucleus. Sections through the rest of the brain stem did not reveal any abnormalities.

Case 2.—A 61-year-old Negro woman was admitted to the Third Neurologic Service of Bellevue Hospital with a vague history of difficulty in speech and weakness of the left lower extremity of three months' duration. One week prior to admission the family noticed that there was weakness of the right lower extremity. Two days later she became markedly "confused" and was unable to walk. According to the family, the patient had always been healthy. They denied knowledge of her having tuberculosis, diabetes, venereal disease, or heart trouble.

General physical examination on admission revealed pulse rate of 74 and regular blood pressure, of 142/80. Chest, heart, and abdomen were normal in all respects.

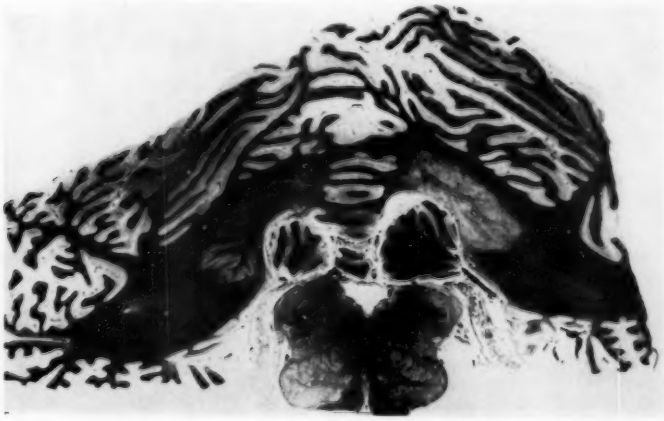


Fig. 1 (Case 1).—Section through the cerebellum and medulla, showing marked demyelination of the right dentate nucleus and right restiform body. The left inferior olive appears larger than the right and shows a slighter degree of demyelination. Myelin-sheath stain; reduced to ⅔ of mag. × 2½.

Neurologic examination revealed the patient to be fully conscious but disoriented and confused. There was evidence of aphasic difficulties. A right facial paresis as part of a right hemiparesis and a right Babinski sign were noted. No visual field defect could be demonstrated. The eyegrounds were normal. The patient was incontinent of urine. X-rays of the skull were normal. X-rays of the chest revealed "interstitial changes and fibrosis of the right lung." There were "enlarged paramediastinal nodes." The spinal fluid was clear and colorless; pressure was normal; the total protein was 48 mg. per 100 cc.; there were no cells; the colloidal gold curve was negative (000000). The Wassermann test was negative. The urine was normal. The blood count revealed a hemoglobin of 14 gm. per 100 cc., with 3,950,000 red cells. The white cell count was 7050, with 68% polymorphonuclears and 32% lymphocytes. The sedimentation rate was 7 mm. in one hour. The NPN was 30 mg. per 100 cc. The blood sugar was 95 mg. per 100 cc. The blood serology was negative. The impression was that of a cerebral disorder, predominantly left-sided, with the probable etiology resting between neoplasm and vascular disease.

Several days after admission rhythmic myoclonic movements of the palate and, to a slighter extent, the posterior pharynx were noted for the first time. The movements were bilateral and regular in rhythm and occurred at approximately 130 a minute. No other abnormal movements of related structures, such as the mouth, the tongue, the larynx, or the eyes, were noted. No abnormal movements of the diaphragm could be observed. Caloric stimulation of both ear drum membranes with cold water elicited strong active responses bilaterally. However, it did not affect the rate or character of the palatal myoclonus. Intravenous amobarbital (Amytal) sodium, 2.5%, given to the point of deep sleep, also did not affect the myoclonus. Carotid sinus pressure also had no effect.

Following pneumoencephalography, 12 days after admission, the patient became comatose. Observation of the palate at this time also did not show any change in the rate and character of the abnormal movements. The pneumoencephalogram revealed slight dilated ventricles but no shift or distortion indicative of an expanding lesion. She died two weeks later, never regaining consciousness. Up to the very day that she died, the myoclonic movements persisted without change in rate or character.

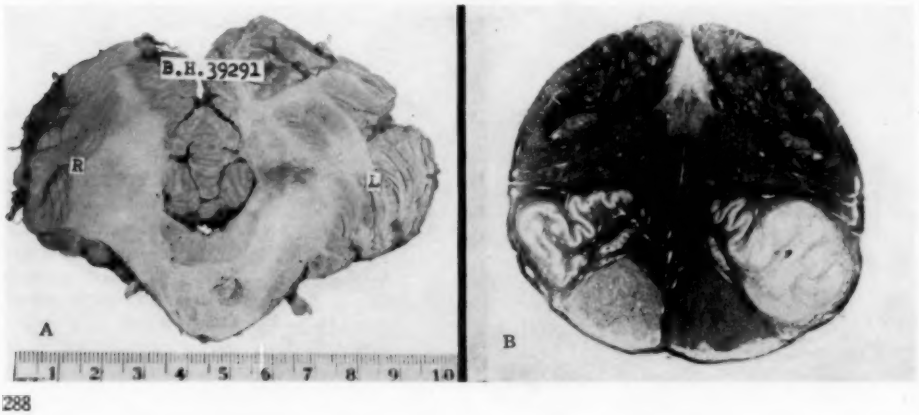
Postmortem examination revealed multiple metastatic carcinoma. Tumor nodules were found in the knee of the corpus callosum on the left, in the left precentral gyrus, and in the right second frontal convolution, and many other, smaller tumor nodules in both hemispheres. Larger nodules also were found in the region of the right dentate nucleus and the base of the pons (Fig. 2*A*). Grossly there was no lesion detectable in the medulla.

Myelin sections of the brain stem and cerebellum revealed destruction of tissue by tumor involving the white matter in the region of the right dentate nucleus. Myelin-stained sections through the medulla revealed a demyelination of the left inferior olive, with the typical "swollen" appearance referred to as "pseudohypertrophy" (Fig. 2*B*).

Case 3.—A girl aged 16 was admitted to the Mount Sinai Hospital with a four-year history of diplopia, headache, and, more recently, vomiting.

Fig. 2 (Case 2).—*A*, section through the pons and cerebellum, showing metastatic lesion in the region of the left dentate nucleus and the basis of the pons.

B, section through lower medulla, showing marked hypertrophy and demyelination of the right inferior olive. There are also some demyelination and beginning hypertrophy of the left inferior olive. Note the demyelination in the left pyramid. Myelin-sheath stain; reduced to 72% of mag. × 7.



Three months prior to admission a posterior fossa exploration at another hospital revealed an inoperable neoplasm, and a Torkildsen procedure was done.

Examination disclosed a bulging suboccipital decompression, secondary optic atrophy bilaterally, horizontal nystagmus on lateral gaze to either side, dysmetria of the left upper and both lower extremities, left Babinski sign, and left hemihypesthesia.

In addition, the palate and pharynx showed bilateral rhythmic myoclonic movements, at approximately 120 per minute. No other related structures appeared to be involved. The nystagmus on both lateral gazes and the pulse rate were not synchronous with the palatopharyngeal movements. There was no noted evidence of its existence prior to this hospitalization. Maintained phonation produced the usual quivering, presumably due to the palatal myoclonus.

Other Observations.—During sleep similar rhythmic movements were seen just beneath the lower jaw near the midline. Palpation of this area and the carotid artery revealed asynchrony. Caloric stimulation again failed to affect the myoclonic movements. Intravenous amobarbital sodium, producing deep sleep, also did not influence the palatopharyngeal movements, although typical lateral gaze nystagmus became apparent.

A ventriculogram revealed a dilated ventricular system down to the upper half of the fourth ventricle, the lower half being seemingly cut off by a mass. A third ventriculostomy was performed. Four months later the patient's condition became critical, and a spinal-subarachnoid-peritoneal anastomosis was done. She died the following day. As the patient's condition progressed and she became comatose, the myoclonus persisted at the same rate and character.

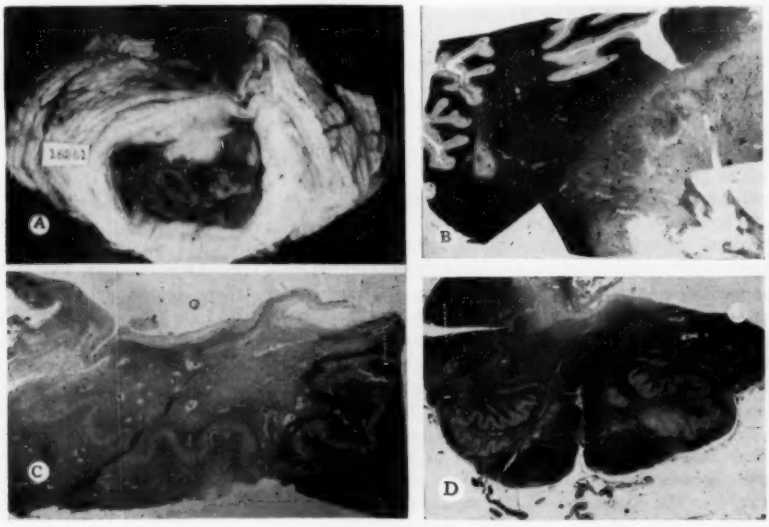
Postmortem examination revealed a tumor (proved to be astrocytoma) in the roof of the fourth ventricle that extended dorsally and laterally to involve the cerebellar hemispheres, more on the right than on the left (Fig. 3*A*). The cerebellar involvement was confirmed on cut sections. Myelin-stained sections from each cerebellar hemisphere revealed a marked destruction of tissue involving the white matter in the region of the right dentate nucleus and, to a less extent, demyelination of the white matter in the region of the left dentate nucleus (Fig. 3*B* and *C*). The myelin sections through to the brain stem revealed a small but definite de-

Fig. 3 (Case 3).—*A*, photograph showing portion of the neoplasm and its extensions laterally, involving the cerebellar hemispheres.

B, section of right cerebellar hemisphere, showing marked destruction and loss of tissue in the region of the dentate nucleus due to tumor. Myelin-sheath stain; reduced to ½ of mag. × 7.

C, section of left cerebellar hemisphere in region of the dentate nucleus, showing demyelination but fairly good preservation of the dentate folia. Myelin-sheath stain; reduced to ½ of mag. × 7.

D, section through medulla, showing hypertrophy and demyelination of a portion of the left inferior olive. This section inadvertently was printed in reverse. Myelin-sheath stain; reduced to ½ of mag. × 7.



myelinating lesion in the left inferior olive, that opposite the cerebellar hemisphere showing the greatest damage (Fig. 3*D*). The rest of the tegmentum of the brain stem and the red nuclei failed to show evidence of a significant lesion.

Case 4.—A 41-year-old woman was admitted to the Mount Sinai Hospital with a history of unsteady gait, incoordination of the left extremities, and occasional double vision for three and one-half years. She was admitted to another hospital, where a ventriculogram showed marked internal hydrocephalus. A suboccipital craniotomy was performed, which showed that the "right cerebellar hemisphere was larger than the left. The folia were widened and discolored slightly yellowish, and along the medial aspect of the right cerebellar hemisphere there was a moderate bulge. A dilated vein ran vertically about 15 mm. from the midline on the surface of the right cerebellar hemisphere. Exploration with a cannula disclosed no evidence of a cyst, but the tissue seemed to have a firm texture. Transcortical incision was made, and several fragments of tissue were removed for study. The vermis was split, and, although the tissue appeared definitely abnormal, some of it was removed for histologic study." Since the operation the patient has developed persistent diplopia, headache, some difficulty in swallowing, and dysarthria. Prior to admission to Mount Sinai Hospital she began to vomit frequently.

Examination revealed bilateral papilledema, coarse horizontal nystagmus on lateral gaze to either side, right sixth nerve paresis, hypesthesia on the left side of the face, and absence of corneal reflexes. There was right facial asymmetry; bilateral decreased auditory acuity; four-limb ataxia, more prominent on the left; hyperactive deep tendon reflexes, and a left Babinski sign.

In addition, for the first time, it was noted that there were abnormal rhythmic involuntary movements of the palate and, to a less extent, of the posterior pharynx and tongue, bilaterally. These movements (140 per minute) were not synchronous with the pulse rate (84 per minute) and were not influenced by phonation (which was quivering) or by carotid sinus pressure. Intravenous amobarbital sodium to the point of sleep also did not affect these abnormal movements, although it did change the character of existing nystagmus on lateral gaze.

A pneumoventriculogram demonstrated a mass in the posterior fossa that extended up to above the third ventricle. A third ventriculostomy was done. When the patient was last seen, two months after this procedure, the myoclonus of the palate, tongue and posterior pharynx was present at the same rate. It is difficult to determine in this case whether the initial operative procedure, in which the cerebellar hemispheres and vermis were cut into, resulted in the production of the myoclonic movements or whether it was due to the existing neoplasm.

Case 5.—A 73-year-old woman was admitted to the Third Neurologic Service of Bellevue Hospital with a two-and-a-half-year history, starting with a motility disorder involving the head and upper extremities, which has persisted. Shortly after the onset of these abnormal movements, her speech became nasal and swallowing became difficult, often resulting in regurgitation of food. She was hospitalized at Medical Center of Jersey City, where a diganosis of Parkinsonism was made. Her condition has been stationary for the past two years.

The most prominent feature of the neurologic examination was rhythmic myoclonic movements of the palate, lower lip, and larynx. The palatal movements were bilateral and appeared to have a circular component. The rate was approximately 140-150 a minute, as compared with the pulse, which was 100. It was apparent that the movements of the lip, larynx, and palate were synchronous. There were no inherent movements of the pharynx or tongue—only slight movements that seemed to be transmitted from the neighboring structures described.

In addition, there were gross rhythmic movements of the head, neck, and upper extremities. These movements varied in rate and character. At times they appeared to be synchronous with the lip, larynx, and palate; at other times dysrhythmic and irregular. On intention there were wild, irregular movements of the upper extremities and exaggerated movements of the head and neck. Occasionally discrete rhythmic movements of the left little finger and the right thumb were noted. Ocular nystagmus was not present, although a cogwheel type of movement on pursuing an object was described by some observers. The tympanic membrane and anal sphincter did not show any movements. Diaphragmatic movements were not synchronous with those of the lip, larynx, and palate. The rest of the neurologic status was essentially normal except for her speech, which was nasal and explosive.

Effect of Intravenous Amobarbital Sodium.—Electrical recording of the movements from the lower lip, larynx, and right hand were made prior to, during, and after the intravenous administration of amobarbital sodium. Immediately after administering 2 grains (0.13 gm.) of amobarbital sodium intravenously, nystagmus of both lateral gazes was noted. These ocular movements became rapid and coarse and obviously not synchronous with the movements of the lip, larynx, and palate, which did not change in rate or character. At the same time as the nystagmus appeared, the movements of the head, neck, and upper extremities stopped. Twenty minutes later the patient fell asleep, and

it was noted that the only movements that persisted were those of the lip, larynx, and palate. Also, the records revealed that during swallowing, yawning, or phonation the movements continued unchanged.

Caloric Stimulation.—Caloric tests produced normal ocular responses but failed to affect the myoclonic movements. Carotid stimulation also did not alter these movements. During natural sleep the lower lip continued to move at the same rate, but the movements of the head and extremities ceased.

CASE 6.—A 59-year-old man gave a four-year history of hypertension and cardiac decompensation, with several cerebral accidents. In one of them he had right hemiparesis, which persisted. Six months prior to admission to the Goldwater Memorial Hospital he had a sudden episode of difficulty in swallowing, slurred speech, and tingling and weakness of the right arm and leg.

Examination showed an enlarged heart and a blood pressure of 200 systolic and 100 diastolic. The patient had frequent outbursts of unprovoked crying and laughing. The pupils were irregular and unequal and reacted sluggishly to light and in accommodation. The gait was reeling. There were dysdiadokokinesis and dysmetria on finger-to-nose testing on the left side. There was a right hemiparesis, with concomitant hyperreflexia and a Babinski sign.

Six months after admission, the patient had another ceerbral accident, accompanied with difficulty in swallowing and slurred speech. It was then noted that there were constant, rhythmic, myoclonic movements of both sides of the palate, posterior pharynx, and larynx. All these movements were synchronous, at the rate of 130 per minute. The palatal movements were not suppressed on phonation. Several months later rhythmic movement of both eyeballs, synchronous with the palatal movements, were also noted. The eye movements consisted of gross rolling of both eyeballs, which moved synchronously, although the excursions of the right eye were of greater amplitude and more rotatory than those of the left. These movements varied with fixation of gaze. When the patient fixed on an object, the rhythmic eye movements, although present, were barely perceptible. However, when the patient closed his eyes, either voluntarily or in sleep, the movements became conspicuous. When the eye movements were first noted by the examiner, the patient complained of double vision, which was present on lateral gaze to either side and was still present when the patient closed either eye. There was no complaint of abnormal movement of objects in the visual field. No weakness of the eye muscles was noted at any time. The complaint of double vision disappeared after several weeks, although the spontaneous rhythmic movements of the eyeballs have continued. There were also rapid, jerking movements of the diaphragm. However, even under fluoroscopic observation, it is difficult to determine whether these diaphragmatic movements were synchronous with the palatal movements.

About six months later, or approximately one year after the palatal movements were first noted, synchronous myoclonic movements of the right lower facial musculature appeared. At this time the following observations were made: The patient appeared dyspneic. Speech was nasal and dysarthric. There were rhythmic movements in the epiglottis, at the rate of about two per second. Associated with these were movements of the palate, tongue, both corners of the mouth, both sides of the diaphragm, and both eyes and eyelids. The eye movements on direct forward gaze were rotatory, showing a clockwise twist. On right lateral gaze the clockwise rotatory ocular movements became more pronounced. On left lateral gaze there was nystagmus with a clockwise component. On upward gaze there were rotatory clockwise ocular movements, more pronounced in the right eye. The same was true on downward gaze and on direct forward gaze. With the eyes closed, the globes were seen to move back and forth in the horizontal plane with a rotatory clockwise component. These movements were quite prominent. The eyelids moved synchronously with the globes. When the eyes were closed tightly, both globes tended to deviate to the left and slightly upward. At the same time the rhythmic clockwise torsion movements of the eyes persisted.

The rhythmic movements described were not directly related to the rhythm of the pulse or to that of respiration. This was shown by simultaneous electrical recording from the eyes, face, larynx, and pulse (Fig. 4).

Effect of Amobarbital Sodium.—It is known that intravenous injections of amobarbital will abolish nystagmus that is present on direct forward gaze. For this reason, we thought that this drug might have a similar effect on the palatal myoclonus. This prediction was not borne out. Under amobarbital sodium narcosis, all the myoclonic movements persisted. Only the rhythmic ocular movements on direct forward gaze were abolished. Neither the lids nor the eyeballs moved when the eyes were open or closed under barbiturate narcosis. This abolition of eye and lid movements appeared within two minutes after 0.23 gm. of amobarbital sodium was given intravenously. This effect persisted for 30 minutes. It should be noted that during this period coarse nystagmus was apparent when the eyes turned in any direction. At no time were the palatal, laryngeal, facial, or diaphragmatic movements abolished or reduced. There was no effect even when the patient was extremely drowsy and did not respond to questions.

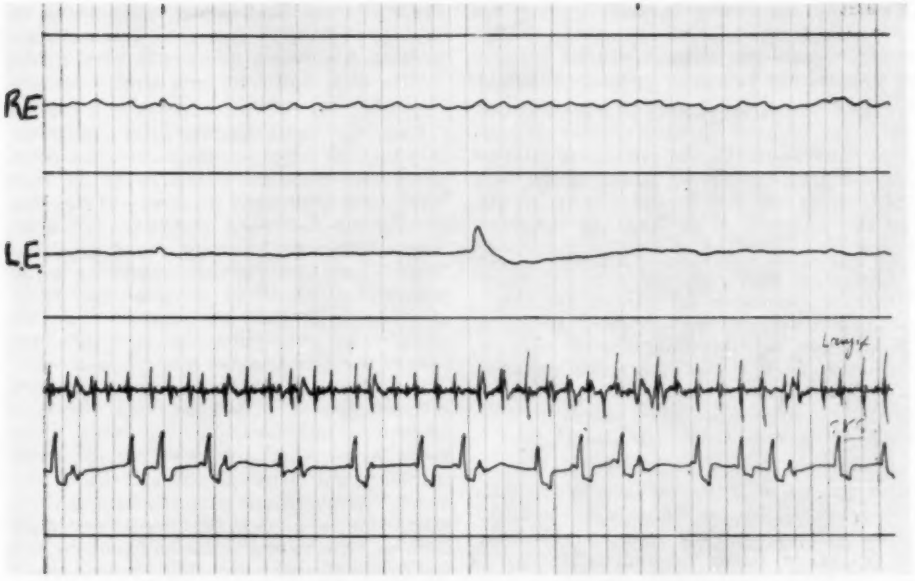


Fig. 4 (Case 6).—Simultaneous electrical recording from the eyes, larynx, and heart beat. This record shows the synchrony of the movements of the eyes and larynx as compared with the slower, irregular heart beat. The movements of the right eye are much more apparent than those of the left eye.

Other Tests.—The patient was given 100%, pure oxygen by means of anesthesia mask for 15 minutes. Neither the palatal nor the ocular movements were altered. Inhalations of 5% CO_2 also failed to affect the myoclonic movements.

The Wassermann reaction of the blood was positive. Lumbar puncture revealed a clear and colorless fluid under an initial pressure of 100 mm. of water. During the manometric readings irregular fluctuations in pressure of up to 200 mm. were apparent. These fluctuations were not synchronous with the palatal rhythm. The spinal fluid contained 10 lymphocytes per cubic millimeter and 87 mg. of protein per 100 cc.; the colloidal gold curve was 1233443200, and the Wassermann reaction negative.

The movements continued at the same rate until his sudden death, four years later (1951). No autopsy was performed.

The diagnosis was hypertensive cardiovascular disease, syphilitic aortitis, aortic insufficiency, and diffuse cerebral and brain stem vascular disease.

Case 7.—A man aged 58 gave a five-year history of unsteady gait. Two years after appearance of this symptom there was a sudden onset of right hemiplegia and aphasia, which cleared in several weeks. During the year prior to admission he noted slurred and halting speech. Examination revealed unsteady gait, circumduction of the right lower extremity, dysmetria of the right limbs, and a bilateral Babinski sign.

There were myoclonic movements of the palate bilaterally. These constant abnormal movements were rhythmic, at a rate of 100 per minute. Related structures, such as the pharynx, larynx, and lip, were not involved. The patient was not aware of these movements, there being no audible click; vocalization when maintained took on rhythmic quiver. The rest of the cranial nerves were intact, except for inconstant fine horizontal nystagmoid jerks on lateral gaze to each side and coarser vertical movements on upward gaze.

Caloric stimulation of both ears (cold water) produced prompt and typical coarse nystagmus, but the rate and character of the palatal movements were unaffected.

Carotid sinus pressure, although it produced a change in pulse rate, failed to influence the myoclonus of the palate.

Intravenous amobarbital sodium (7½ grains [0.5 gm.] in 2.5% solution) to the point of deep sleep also did not alter the characteristics of the palatal myoclonus. As expected, there was slowing of respiration and pulse rate, in addition to the barbiturate nystagmus.

Complete work-up at another hospital, including a pneumoencephalogram, was negative. The history of right hemiplegia and aphasia suggested that more than one lesion existed, and therefore he was suspected of having multiple sclerosis.

CASE 8.—A man aged 49 was admitted to the Mount Sinai Hospital with a two-year history of unsteady gait, occipital headache, and nausea. There was a background history of hypertension of at least 10 years' duration.

Examination revealed a blood pressure of 300/110. There was a right facial paresis. Auditory acuity on the right was decreased. Dystaxia of the left extremities was prominent. Eyegrounds showed hypertensive retinopathy. Abnormal rhythmic movements of the palate and posterior wall of the pharynx, at a rate of 120 per minute, were observed. The pulse rate was 84 per minute. Phonation was nasal and quivering in character. No other related structures were involved.

Caloric stimulation bilaterally produced active ocular responses but had no effect on the myoclonus. No effect was noted with carotid stimulation. Intravenous amobarbital sodium, producing sleep, also had no effect, although it produced typical barbiturate nystagmus on lateral gaze to either side.

The impression was that of a posterior fossa disorder, probably vascular in origin, all laboratory work-up for expanding lesion being negative.

CASE 9.—A 55-year-old man was admitted to Goldwater Memorial Hospital on June 15, 1950, with a two-year history of a "stroke" involving the left extremities. This was followed several months later by a gradual onset of ataxia, incoordination, tremor of the extremities, and dysarthria. For a period during this time the patient complained of "double vision."

Neurologic examination revealed bilateral sixth nerve palsy and horizontal nystagmus of the left eye on attempted left lateral gaze. There was a pronounced tremor of the left extremities, which, on intention, became gross wing-beating movements. At times there were involuntary flinging movements of the left extremities, resembling hemiballismus. A left Babinski sign was present.

In addition to these findings, there were bilateral abnormal involuntary movements of the palate at the rate of 100 per minute. To a less extent, the tongue and pharynx were also involved. Phonation, natural sleep, carotid sinus pressure, caloric stimulation, and intravenous amobarbital sodium did not alter these abnormal movements in any respect. However, the intravenous barbiturates did abolish the abnormal movements described in the left extremities. All laboratory procedures, including lumbar puncture, were within normal limits. These same abnormal movements have been observed without change since 1950.

The impression was posterior fossa disorder of undetermined etiology.†

† Since this paper was submitted, further brain stem and cerebral changes developed in this case, and the palatal myoclonus stopped.

CASE 10.—A 66-year-old woman was admitted to the hospital with a history of sudden loss of consciousness, resulting in a fall down stairs and fracture of the skull, two years before. The cerebrospinal fluid was found to be bloody. Upon her regaining consciousness, a severe left hemiparesis was noted. Several weeks later it was noted that her speech was dysarthric, and there was incoordination of the right extremities. She was admitted to the Third Neurologic Service of Goldwater Memorial Hospital on June 10, 1952.

There was a background history of severe hypertension. Examination revealed her blood pressure to be 200/100. In addition to a left spastic hemiparesis, there was dystaxia of the right extremities.

Pronounced rhythmic abnormal involuntary movements of the lips, tongue, pharynx, and larynx were observed. Her speech was almost unintelligible and had a jerking, quivering quality. The movements of all the structures described were synchronous and at approximately 132 a minute. Observations made during natural sleep, carotid sinus pressure, caloric stimulation, and intravenous administration of amobarbital sodium failed to show any significant effect on the rate and character of these movements. Fluoroscopic studies showed movements of the diaphragm that were thought to be synchronous with the movements of the palate, tongue, etc.

Further neurologic work-up did not suggest an expanding lesion in the posterior fossa. The patient was discharged in July, 1953, with no change in her status. The impression was vascular disease involving the structures of the posterior fossa.

CASE 11.—A 59-year-old Negro woman was admitted to the Mount Sinai Hospital with a three-year history of unsteady gait with veering to the left, vertigo, diplopia, and slurred speech, which had gradually become worse in the past year.

Neurologic examination revealed horizontal nystagmus on lateral gaze to either side and vertical nystagmus on upward gaze. There were gross myoclonic movements of the palate and posterior pharynx, at approximately 110 per minute. Her speech was slurred, and on phonation there was a definite quiver of her voice. There was a marked unsteady gait and dysmetria of all four extremities. Deep tendon reflexes were hyperactive throughout, with bilateral Hoffman and Babinski signs. Laboratory procedures, including x-ray examination of the skull and lumbar puncture, were normal. The impression was brain stem disease of undetermined etiology, probably degenerative.

During natural sleep the rhythmic myoclonic movements could be palpated in the neck; these were not synchronous with the heart beat. Carotid sinus pressure failed to change their rate and character. Caloric stimulation of both ears produced changes in the existing nystagmus but did not alter

the palatal movements. Intravenous amobarbital sodium to the point of deep sleep failed to affect these movements in any way.

This patient has been followed in the clinic for the past year, and the myoclonic movements persist.

CASE 12.—A 49-year-old woman was admitted to the Mount Sinai Hospital with a four-year history of a left hemiplegia with moderate improvement. Shortly after the onset of this episode she began to notice that the left eye turned inward and that her speech became dysarthric. One month prior to admission she began to complain of nausea, vertigo, and, at times, diplopia. There was a background history of severe hypertension, of 15 years' duration.

Neurologic examination revealed a left sixth nerve palsy and an oblique horizontal nystagmus on right lateral and direct forward gazes with accompanying oscillopsia. There was a residual left hemiparesis and left Babinski sign. In addition, there were rhythmic abnormal movements of the palate and pharynx bilaterally, at approximately 180 per minute. Phonation had no effect on these movements. Carotid sinus pressure and caloric stimulation and intravenous barbiturates to the point of deep sleep all failed to influence the palatopharyngeal movements.

Laboratory work-up failed to reveal any evidence of an expanding lesion in the posterior fossa. The blood pressure in the hospital remained elevated 260-280/120. The impression was (1) posterior fossa disorder, probably vascular, and (2) residual left hemiparesis secondary to a vascular lesion in the right cerebrum.

COMMENT

Clinical Characteristics.—It appears under the variety of conditions studied that myoclonic movements of the palate and related structures, once they occur, persist, with no definite change in rate or character, for the rest of the subject's life. Their resistance to influence by drugs, sleep (natural and induced), caloric stimulation, CO_2 inhalation, coma, impending death, and carotid sinus pressure makes these abnormal movements unique and sets them apart from other movements, normal or abnormal, voluntary or involuntary. Even such regulated, automatic, centrally controlled movements as the heart beat and respiration fluctuate under certain conditions. Dyskinesias, such as tremors, chorea, athetosis, hemiballismus, and nystagmus on direct forward gaze, have been shown ‡ to be affected (abolished) by intravenous amobarbital and natural sleep, whereas the myoclonic movements of the palate and related structures were not affected. This is well illustrated in Case 7, in which palatal myoclonus existed with the tremor of Parkinson's disease of the extremities. Intravenous amobarbital promptly stopped the tremor but did not influence the palatal movements. The only movements that may be comparable are fasciculations (fibrillations) due to anterior horn cell disease. Intravenous barbiturates also fail to influence these movements.

The term myoclonus is a descriptive one, often used but rarely defined. Although abnormal movements in different persons may look alike, they may not act alike under certain conditions. Recently, this point was brought out by Bergman, Nathanson, and Bender,[9] who described two patients with visibly identical abnormal gross jerky movements of the muscles of the buttocks, thigh, and abdominal wall. In one patient intravenous amobarbital failed to alter these movements. The "myoclonus" of the other patient was promptly abolished by intravenous amobarbital. An electroencephalogram in the latter case revealed synchronous spikes, suggesting that the abnormal muscle movements were cerebral in origin. Several days later, the movements stopped spontaneously, and the electroencephalogram no longer showed spike activity.

Further investigation along this line, especially the effect of drugs on abnormal movements, is indicated and should help classify and eliminate assumptions made from naked-eye observations alone.

Pathophysiology.—The three postmortem studies give strong evidence that the major and primary lesion was in the cerebellum, with the greatest destruction of tissue in and around one of the dentate nuclei. These lesions were associated with demyelination in the opposite inferior olive. In two of the cases the major cerebellar lesion was neoplastic, thus allowing the safe presumption that the tumor existed before the demyelinating lesions in the opposite inferior olive. Two

‡ References 9-13.

surgical cases of posterior fossa tumor associated with palatal myoclonus reported by Erickson and Ablin [14] also suggest that the primary site producing this phenomenon is other than the inferior olive.

The data presented, both from autopsy and from anatomic localization at operation, although gross and presumptive, indicates that the major pathology originated in one cerebellar hemisphere and in time caused retrograde degeneration of the opposite inferior olive. The resulting dysfunction of this connecting system seems necessary to set off the phenomenon of myoclonus.

Experimentally, Weinstein and Bender [15] elicited myoclonic movements of the palate, tongue, lips, etc. by electrical stimulation in the region of the hilus of one inferior olive in the monkey. Recently in preliminary investigations by Bender and Teng, a destructive lesion in the region of one inferior olive of a monkey produced typical persistent myoclonic movements of the palate. Natural and induced sleep (intravenous barbiturates) failed to alter these movements. Plans are under way to make destructive lesions in one cerebellar hemisphere in the monkey and to observe the animal for possible development of palatal myoclonus. An interesting, and perhaps pertinent, observation was made by Harmark [16] in a study seemingly unrelated to this problem. Harmark investigated the influence of the cerebellum on distant cell migrations from the rhombic lip to the inferior olive in chick embryos. He found that when the cerebellar anlage was injured in the embryos of 13 days or older, invariably there was secondary alteration in the region of the opposite inferior olive. Unfortunately, observations as to abnormal involuntary movements were not made in those chicks that were allowed to hatch.

There is little question that the inferior olive is one of the most important links in the anatomic system for setting off these abnormal movements. However, it should be emphasized that lesions elsewhere (particularly in the opposite cerebellar hemisphere) are commonly the anlage responsible for the secondary changes in the inferior olive.

SUMMARY

A study was made of 12 patients showing the phenomenon of myoclonic movements of the palate and related structures. Clinical observations under a variety of conditions revealed that these abnormal movements were unique in that they were not influenced by natural sleep, phonation, intravenous barbiturates (induced sleep), carotid sinus pressure, caloric stimulation, coma, and impending death.

The pathophysiology based on three of the four patients who died is illustrated and discussed. The data revealed that the primary site of pathology occurred in one cerebellar hemisphere and was associated with secondary retrograde changes in the opposite inferior olive. In another case, there developed myoclonus of the palate three months following removal of a posterior fossa tumor with excision of part of the cerebellar hemispheres.

It behooves the clinician to investigate the existence of cerebellar hemisphere pathology when he encounters myoclonus of the palate, rather than to consider it to be entirely the result of a lesion of the inferior olivary nucleus.

Dr. Lewis D. Stevenson, neuropathologist, Bellevue Hospital, made the pathologic study in Cases 1 and 2, and Dr. Irwin Feigein, Mount Sinai Hospital, New York, in Case 3.

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Special Articles

SOME OBSERVATIONS ON THE RELATIONSHIP BETWEEN PSYCHIATRY AND THE LAW

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On the assumption that mental states and impulses of a certain kind are caused only by "disease" or "sin," two equally vague conceptions in this context, doctors are asked to discriminate nicely between the two, and they naturally tend to flounder badly in the process. The underlying theory appears to be that the law permits some modes of conduct to be caused—e. g., by disease—but not others. The latter are said to be the product of free will, which is omnipotent enough to create the first link in a chain of thought or conduct. By accepting a particularly arbitrary distinction between mental health and disease, doctors pass beyond the facts of their own science, and by accepting the legal view of free will they abandon the only fundamental canon of all science.

ERNEST JONES [34]

INTRODUCTION

In these troubled times, many psychiatrists, lawyers, and intelligent lay persons share an intense interest in the question, "What can modern psychiatry contribute to our understanding of the psychology of antisocial behavior."

In this essay I propose to examine certain concepts and phenomena which play a central role in present-day discussions of the psychological aspects of criminology. It is my impression that most workers interested in this field are rather strongly driven by a wish to make some practical suggestions for modifying current practices regarding the orientation which the law should take toward the offender. I believe that such a frame of mind is not conducive to scientific progress in the realm of fundamental concepts. Moreover, since any consideration of our subject brings us into contact with notions such as "mental health and disease," "the ability to distinguish between right and wrong," "free will," "criminal responsibility," and a host of others, it seems to me worth while to devote some attention to a theoretical examination of such concepts. In other words, I shall propose no changes in the existing rules governing criminal law. This implies neither satisfaction nor dissatisfaction with the present state of affairs in this realm of human living. It implies only a desire to understand a problem which, in large part, is a psychological one.* Accordingly, the purpose of this essay is to explore and, if possible, to clarify the nature of our inquiry regarding the relationship between psychiatry and the law.

It is all too often assumed that so-called "knowledge," irrespective of its source of origin or function, can be usefully applied to problems in areas of inquiry other than the one in which the "knowledge" to be applied arose. While at times this may be possible, at other times such ventures mislead the investigator. The application of psychiatry to the law, for example, disregards the important differences in the respective frames of reference in which human behavior is viewed by these two disciplines. Robert Waelder commented about this,† albeit in a context unrelated to our present subject, as follows:

> The difference which exists between our attitude in social relationships and a pedagogic or therapeutic attitude amounts to this, that in the former we treat our fellow-men as if they were completely free; we make demands, appraise and condemn. As pedagogues and therapists we treat the other party,

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* That is, if by the study of psychology we understand quite broadly all those processes which concern man's relationship to himself and others.

† Waelder,[55] p. 103.

or more correctly, the object of our pedagogical or therapeutic activity, as one who is not free; or, to be more precise, as though his freedom were limited in the several ways we have described. Why indeed this should be the case, why man's social relationships require him to treat others as though their conduct were entirely free—failing this, the social relation is disturbed—would need a separate investigation.

Waelder's comments about the nature of "social relationships" apply, of course, to the psychology of the law. On the other hand, we take his comments about the "pedagogic or therapeutic attitude" also as a statement of the position of psychiatry as a scientific theory vis-à-vis the problem of human relationships. One of the aims of this essay is thus to investigate the aforementioned question posed by Waelder, in which he asks why it is that in social relationships we treat our fellow men as if they were completely "free" to do as they please and, accordingly, hold them "responsible" (with certain important modifications and exceptions) for their actions.

CRITICAL SURVEY OF PSYCHIATRIC POSITIONS REGARDING CONTRIBUTIONS OF PSYCHIATRY TO THE LAW

I believe it is fair to say that currently there is a widespread conviction among psychiatrists that on the basis of our knowledge of human behavior we could make valuable, humanistic suggestions regarding the legal aspects of criminology.‡ I will try to show that this does not follow necessarily or inevitably from the premise that we do have a better scientific understanding of behavior than we did 50 years ago. Scientific knowledge per se does not embody within itself any guideposts as to its "best" or "most humanitarian" utilization, much as we would like to believe this. We know this only too well from our experiences with dynamite and nuclear energy. Nevertheless, it seems to me that there is an implicit belief in the minds of most psychiatrists interested in legal matters that the application of psychiatric knowledge to jurisprudence is bound to make the law more "humane" and "just," rather than less so. This assumption should not be accepted at face value. We should explicitly entertain the possibility that changes in the law may also lead to results which may be regarded by some as not altogether desirable.§

It may be noted in this connection that Zilboorg, who has probably made more enduring contributions to the psychological aspects of criminology than any other psychiatrist of our time, has recently warned us of just such a trend in psychiatry in general. He expressed the belief that modern psychiatry has already moved away from "humanism in its true sense [which] is served poorly or not at all when the individual becomes but a psychobiological adaptive molecule of society, when the individual seems to be looked upon as having been born to serve society, instead of society's having been developed to serve man."[61] Similarly, Jones noted that in our time the social ideal of "loyalty" has displaced the 19th century ideal of "freedom."[35]

Guttmacher is one of the strongest advocates of legal changes regarding "criminal responsibility." His position is characterized by a criticism of the M'Naghten Rules and by an advocacy of the concept of "mental illness" as a *cause* of the criminal act. In a recent paper, Guttmacher pleads for a more general acceptance of the New Hampshire Rule, which "rejects all legal tests that have been devised and holds that the question of irresponsibility is one of fact for the jury. Under this law, if the defendant has a mental disease and if the criminal act is the product of it, he is found not guilty by reason of insanity. The fundamental principle behind this is that criminal responsibility requires

‡ No attempt is made in this essay to present a systematic review of the literature on the relationship between psychiatry and the law. The contributions of Guttmacher (References 30 and 31), Overholser,[42] Weihofen,[56] Wertham (References 57 and 58), and Zilboorg (References 60 and 62) are among the most important ones representative of the current thought in this field. The reader's attention is also called to the recent report on this subject by the Group for the Advancement of Psychiatry.[29]

§ References 40 and 50.

guilty intent, or *mens rea,* as well as a prohibited act." ‖

Guttmacher makes the following concrete suggestions [30]:

> It seems to me that all that should be expected of the psychiatrist is the following:
>
> 1. A statement as to whether the defendant is suffering from a *definite and generally recognized mental disorder* and why and how this conclusion was reached. (Italics mine)
>
> 2. If it has been asserted that the defendant suffered from a mental disorder, its name and its chief characteristics and symptoms, with particular emphasis on its effect on an individual's judgment, social behavior, and self-control, should be given.
>
> 3. There should then follow a statement of the way and degree in which the malady has affected the particular defendant's behavior, especially in regard to his judgment, social behavior and self-control.
>
> 4. He should then be asked whether the alleged criminal act was, in his opinion, a product of mental disorder.

It is important to note that the foregoing suggestions may or may not lead to socially desirable changes in the practice of criminology. In either case, they cannot be supported by claims of having been derived from psychiatry as a science of human behavior, for the following two reasons:

1. The notion of a "generally recognized mental disorder" denies the basic scientific fact that people possessing diverse life experiences (i. e., differing one from another in skill, education, culture, etc.) have different notions of what constitutes "mental illness." Accordingly, the conception of a "generally recognized mental disorder" must be recognized to represent a statement which discloses a significant uniformity of life experiences among the observers; it is devoid of meaning regarding the alleged phenomena of observation (i. e., the "patient").¶

2. There is a logical fallacy in attributing some modes of behavior to "disease" and others to "free will." [34] The notion of "causality" is misinterpreted and is used to dissipate the painful feeling of human "responsibility." We shall return to this problem later.

Overholser, too, advocates the application of psychiatric "knowledge"—resting squarely on the distinction between mental health and disease—to judicial change in regard to the offender. He states [42]:

> We may safely expect that with the development of mutual understanding between representatives of the law and psychiatry the adoption of the needed improvements which we have discussed may be accelerated.#

‖ Reference 30. It is of interest to add that the above was one of five proposals for psychiatric changes regarding the legal handling of criminals, presented by the author in his paper addressed to the annual meeting of the American Psychiatric Association (1954). Eighty-five per cent of those who heard the paper voted in favor of this proposal.

¶ In 1665 Sir Thomas Browne, one of the most distinguished surgeons of the time, testified in court that witches did indeed exist, "as everyone knew" [42] (p. 106). Today, every learned physician "knows" that there "exist diseases of the mind," just as there are diseases of the body. Similarly, "everyone knows" that there is such a thing as "responsibility," and, since it has to do with the mind, psychiatrists ought to know more about it than other people. In this connection, see Bowman and Rose [9] and Brill and Morse, in discussion on their paper, pp. 406 and 408, respectively.

Overholser,[42] p. 134. Elsewhere, Overholser states: "When, however, the question of mental condition is raised by a defendant (as it must be in a criminal prosecution) it seems to me at least that he has unsealed the lips of the physician." [41] Accordingly, Overholser believes that the physician may and should testify either for or against the defendant. From the point of view of scientific psychology, one must, of course, ask why a physician wishes to testify in a court of law. What prompts him in his desire to testify, and what conscious and unconscious gratifications does he derive from so doing? The desire to gain power over men must be mentioned in this connection as an unquestionably relevant consideration.[47] The assumption that the power motive is of importance to the physician who likes to transfer his activity from the office into the courtroom is supported by the rarity with which psychiatrists interested in legal matters suggest that the specialist in this field should abstain from testifying altogether. Yet, this inference seems to follow logically from the fundamental differences in the respective orientations of law and psychiatry to human behavior. I believe that the conviction to *not* testify in any manner whatsoever is quite widespread among practicing analysts; this, however, does not get into print, and perhaps contributes heavily to the distorted impression which one might gain from publications regarding the role of the psychiatrist in judicial proceedings.

Overholser's position, it seems to me, disregards completely the historical fact that increasing specialization in diverse fields of inquiry tends to lead to less, rather than to more, "understanding" among workers in different fields. Thus, to take as an example our present concern with medicine, psychiatry, and law, it would seem justifiable to maintain that there was much harmony among these disciplines in ancient times, when the roles of doctor, psychiatrist, and judge (and priest) were united in a single person. The notion that "increased knowledge leads to better understanding," even if different types of "knowledge" are involved (as in the case of psychiatry and law), represents a sentimental rather than a scientific view of the social function and results of "science."

The significant differences in the positions which psychiatry and the law take vis-à-vis the problem of human behavior are brought out beautifully by the words of Sir James Stephen (written in 1863), who is quoted by Overholser, but with whose views the latter disagrees.

> Does then the law affirm any, and if so, what systems of morals be true? The law makes no such affirmation. It has nothing whatever to do with truth. It is an exclusively practical system invented and maintained for an actually existing state of society.*

I shall present a fuller discussion of the complementary,† rather than additive, character of the legal and psychiatric views of human conduct in subsequent sections of this essay.

The report of the Group for the Advancement of Psychiatry, entitled "Criminal Responsibility and Psychiatric Expert Testimony," [29] is also characterized by an emphasis on the concept of "mental disorder" and so-called causal connections between disordered mental states and criminal acts. In this report the suggestion is also made that sentence be deferred "in every case in which the question of mental disorder is raised.‡

A recent editorial in the journal *Psychiatry* adds to the consistency of the foregoing point of view regarding criminal responsibility. Here, too, the notion of "mental illness" is emphasized and is regarded as a cause of the prohibited act.[13]

Revisions of the criteria of "responsibility" (and punishability) according to these concepts were considered by committees of jurors and psychiatrists in the District of Columbia during the fall months of 1954. These were reported in articles and editorials in the Washington papers. In one of these, entitled "Need of New Insanity Laws to be Explored," [38] it was stated:

> A number of suggestions already have been made for changes in the law or for new facilities. These include:
>
> A diagnostic center where top psychiatrists can sort out the mentally ill from the malingerers and make a more complete study of the defendants than can now be done in brief visits to District Jail.
>
> A law to enable mental hospitals to hold defendants not only until they are considered of sound mind, but also until they are considered to be safe at large. A law spelling out a definition of insanity.

Let us examine briefly the alleged sources of evidence upon which these views are based. In this connection, I want to call special attention to the many errors to which one is exposed in "applying" theoretical knowledge to everyday living, and particularly to matters of ethical judgment.

Psychiatric expert testimony and the notion of "mental illness" are based on the concept that psychiatry is the science of human feeling, thought, and action. Mathematics and physics are also sciences; in fact, they are our models of what science *is*. Yet, while there may be agreement among scientists as to their theoretical (i. e., scientific) knowledge, it does not follow that there is agreement among them as to *what to do with it*.§

It is common knowledge that, while scientists may share the same theoretical

* Overholser,[42] pp. 40-41.

† References 7 and 8.

‡ References 29, p. 7.

§ We have witnessed a rather dramatic illustration of this in connection with the recent "investigation" of Dr. J. Robert Oppenheimer.[53]

views regarding their "science," they may differ widely on how they "apply" this knowledge to their religious and political conduct. Thus, scientists, like other citizens, have contributed their knowledge, sometimes toward furthering the interests of the prevailing social order and at other times in opposition to it. Now, it may be argued that this example is far-fetched, that there is a great gap between theoretical physics and one's political conduct. That is true. Yet, what I want to emphasize is that there is always a gap between knowledge which is aimed at *understanding* (science), on the one hand, and knowledge aimed at justifying or bringing about *personal or social change*, on the other.‖

Let us return to psychiatry and see how this applies. Modern psychiatry is said to consist of a body of knowledge upon which there is more or less general agreement. This knowledge consists of, or is derived from, the theory and practice of psychoanalysis. Few psychoanalysts will argue about such a definition of modern psychiatry, provided it is kept on the level of a theory, that is, a system of thought which aims at explaining certain phenomena. As soon as we come to the application of these thoughts, even to psychotherapy, disagreement begins. Illustrative of this principle are the divergences of opinion about therapeutic technique even among analysts, who otherwise share a large body of common theory. Does it not follow, then, that when we propose to apply psychiatry to social change the relevance of the alleged theoretical foundations for change rests on extremely shaky ground? Theory now becomes a matter of "interpretation," with the result that each term and concept acquire a different meaning for different persons, and even for the same person at different times.

A survey of the psychoanalytic literature will show that the propositions currently put forward in the name of psychoanalysis are by no means the only ones that have been derived from this source. A review of the older literature discloses the interesting fact that neither Freud nor the other founders of this science, including Abraham, Ferenczi, and Jones, seem to have paid much attention to the question of "criminal responsibility."

In a paper published in 1906, Freud had this to say [22]:

> The task of the therapeutist is, however, the same as the task of the judge; he must discover the hidden psychic material. To do this we have invented various methods of detection, some of which lawyers are now going to imitate.¶

Few psychiatrists or jurists would agree with this statement today. Nor is it possible to imagine that Freud could have written this in 1938. In addition to its being a reflection of certain cultural features of the spirit of "freedom" and "truth" which permeated Europe in the decades before the First World War, this statement also betrays Freud's characteristic scientific orientation in his emphasis on the search for "what really happened." Indeed, the title of the paper from which this passage is quoted is "Psycho-Analysis and the Ascertaining of Truth in Courts of Law." Thus, for the sake of making his point about psychoanalysis, Freud chose to stress the similarities between it and certain purposes of the law. At the same time, however, he did not dwell on the differences between the two, which most of us would regard as outweighing the similarities.

Ferenczi's writings on this subject were aimed, on the whole, on acquainting lawyers

‖ In connection with these considerations, I want to mention Bertrand Russell's important contributions in "Religion and Science" to many problems closely allied to those discussed in this essay. Considering the connections between science and ethics, Russell wrote: [48] "Questions as to 'values'—that is to say, as to what is good or bad on its own account, independently of its effects—lie outside the domain of science, as the defenders of religion emphatically assert. I think that in this they are right, but I draw the further conclusion, which they do not draw, that questions as to 'values' lie wholly outside the domain of knowledge. That is to say, when we assert that this or that has 'value,' we are giving expression to our own emotions, not to a fact which would still be true if our personal feelings were different" (p. 242).

¶ Freud,[22] p. 18.

with the discoveries of psychoanalysis, as a theory of instinctual (motivational) behavior.# Neither he nor Freud seemed to have seriously entertained the idea that a person should not be punished by the law because of what psychoanalysis may tell us about him —a startling fact in the light of current thought on this matter.

The assertion that Ferenczi did not advocate any modifications in what we now call "criminal responsibility" is borne out by the following statement [21]:

> A condemned person has no further reason for concealing anything of the thoughts and associations by means of which the unconscious motives for his actions and tendencies may be brought to light. Once the treatment has been begun, then the so-called "transference," the emotional bond with the person of the analyst, will even render him desirous of and pleased with being dealt with in this way. The comparative investigation of similar offences will then render it possible to fill in the gaping lacunae of criminological determinism with solid scientific material.*

Note again, first, the assumption that psychoanalytic knowledge is being applied after conviction, and not in order to modify the sentence, and, second, the emphasis on scientific inquiry. The contrast between this view and the present emphasis on bringing about legal changes, with only perfunctory appeal to "research," is striking. It is consistent with this changed attitude that little has been added to our knowledge of the psychology of antisocial behavior by those who advocate legal changes. The advances in this area which have been achieved were largely through the efforts of workers who have not expressed themselves directly on the subject of legal modifications for the handling of offenders.†

Finally, Jones [24] has offered us one of the most lucid arguments that can be brought against the naïve approach of advocating legal changes on the basis of the notion of "mental illness." The passage most relevant to our theme is quoted at the beginning of this essay. In it, Jones pinpoints the error inherent in a view which regards some modes of conduct as due to "disease" and ascribes others to "free will." ‡

WHAT IS "MENTAL DISEASE"?

The foregoing considerations lead us logically to consider the questions "What is mental disease?" and "What is mental health?"

Historical considerations are helpful in this connection, for they show that during those times when the study of man's behavior fell into the domain of the religions, variations in such behavior were described as "sinful" or "virtuous" (or bad and good), while later, when these phenomena were taken over by medicine, they acquired the new labels of "sick" and "healthy." Since modern psychiatry originates from medical sources (Freud and his teachers, Kraepelin, Bleuler, Meyer, etc.), it is not surprising that the early pioneers in psychiatry were strong proponents of the view that certain modes of behavior constitute "illness." Indeed, in their times the acceptance of such a view was a legitimate goal and represented a genuine advance over the belief that aberrant behavior—unless "due to brain disease" —was either sinful or meaningless (i. e., "just crazy"). Thus, Freud patterned his thinking strongly along the lines of organic pathology and built up a system of "psychopathology." Moreover, the wish to have nonpsychiatric people look upon aberrant behavior as an "illness" was an important matter in Freud's day, as we well know. This attitude has been

References 20 and 21.

* Reference 21, p. 436.

† 1, 2, 19, 23, 25, 33, 51.

‡ For an excellent discussion of the relationships between the concepts of volition and cause, see Russell's "On the Notion of Cause, with Applications to the Free Will Problem." [46] It seems to me that to undertake the application of psychiatric knowledge to social change without a thorough understanding of the philosophical issues involved in these problems will bring disaster upon us just as surely as did the use of x-rays bring disaster upon the medical men who first used radiation without the proper precautions.

carried over to our present era, and even today there seems to be a belief among psychiatrists that by convincing the laity (and our medical colleagues) that there is such a thing as "mental illness" a great intellectual and humanitarian advance will have been wrought.

In contrast to this view, it is my thesis that the model of organic medicine (and of health and disease) has served its purpose for psychiatry and that it has now become a hindrance rather than a help to further advances in our field.

There are two somewhat different directions from which the validity of the foregoing assertion can be substantiated: First, by showing how this analogic model has served its intended purpose, and, second, by calling attention to its important weaknesses, that is, by a shift of emphasis from the common to the distinguishing features of psychiatry and medicine.

The origin of psychiatry from neurology and medicine was one of the reasons for trying to order behavior according to the concepts of "normal" and "abnormal." Since psychiatry was not "accepted" until recently, in the sense of social approval, the emphasis on the part of psychiatrists that their work was similar to that of medicine can be further understood as a legitimate social maneuver to improve their position. Clearly, in this function the medical model has served psychiatry well. However, I think if we agree that this is one of the bases for the evolution of the notion of "mental illness," then we must agree that this notion is simply the outcome of the use to which the model of organic medicine was put by psychiatry; and it then remains to be shown that there are, in fact, significant similarities between the methods and conceptual frameworks of medicine and psychiatry which would warrant the retention of this concept.

An examination of this latter issue could well form the subject of a lengthy treatise. However, I wish to be brief, and to those who see the matter in a different light my thesis may well seem arbitrary.§ I submit that the methods and conceptual framework of psychoanalaysis and of modern psychiatry are significantly different from the methods and concepts of organic medicine. The most crucial difference seems to lie in the following: In organic medicine, we have certain criteria by which many people of widely varying backgrounds and beliefs can agree on what constitutes health and disease. This follows simply from the fact that the function of the body as a machine is given in large measure by the way its parts are put together and work. Accordingly, anything which interferes with life (i. e., growth, reproduction, etc.) is regarded as "unhealthy"; and, contrarily, the state in which there is no interference with the workings of the bodily machinery is called "health." The crux of the matter lies in the *general agreement* that the "body is for living," so to speak. It is relevant to note that under certain cultural conditions there may be a disagreement as to this definition of health, and then we see an attitude toward organic illness at variance with our present point of view.|| The notion that the function and "purpose" of the body is an invariant factor is well illustrated by a quotation from Judge Doe, writtent in 1866, and cited by Guttmacher:

> The law does not define disease—disease is so simple an expression that the law need go no further. What is a diseased condition of mind is to be settled by science and not by law—disease is wholly within the realm of natural law or law of nature.[30]

Today, most of us no longer speak of "natural law," any more than we think in terms of "original sin." We conceive of all laws, scientific as well as judiciary, as man-made. Where does this leave us with the question of "mental disease."

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§ For a detailed discussion of this subject, see my paper presented at the Annual Meeting of the American Psychoanalytic Association, Atlantic City, N. J., May 7, 1955, entitled "On the Theory of Psychoanalytic Treatment," to be published.

|| For example, seeking "eternal life" through martyrdom in early Christianity.

We have seen that organic disease is a useful concept because of the wide agreement among people concerning the nature of the function and purpose of the human body. If psychiatry is a science of human behavior, obviously we would have to have a comparable level of agreement as to the function and "purpose" of human conduct before a relevant definition of "mental disease" could be reached. We have no such agreement in this matter. To the extent to which there is agreement, we speak of societies, cultures, groups of friends, scientific bodies, and so on.

Accordingly, it seems to me that we are forced to conclude that we cannot define "mental health" in the same way as we can physical health. Whenever we try to give a definition of what mental health is, we simply state our preference for a certain type of cultural, social, and ethical order; and, on the contrary, we designate deviations from such an explicitly stated, or more often hidden, model as "mental disease." As scientists, we have no choice but to accept the limitations, as well as the accomplishments, of what science can do.

Is there a more positive contribution that psychoanalysis can make to the problem of "health" and "normality"? In regard to this question, I find myself in agreement with a point of view expressed by Eissler,[18] who has recently suggested a definition of what may be called mental health according to the ego's response to, and ability to cooperate with, the unmodified technique of psychoanalysis. This has all the earmarks of a clear and useful definition. At the same time, however, by this definition the concept of mental health is taken out of the "practical" context of actual cultural conditions and legal conventions.

WHAT IS "RESPONSIBILITY"?

The word "responsible" is used in everyday language in at least two distinctly different ways. One of these relates to our notions of cause and effect. For example, we say that "the avalanche at X was *responsible* for the death of three skiers." This simply means that we draw a causal connection between the avalanche and the death of the skiers. It is a statement of *what happened*. It does not contain any (direct) evaluation of the ethical quality (good or bad) of the event or any suggestion about what should be done. In contrast to this, the second meaning, in which the word "responsible" is used—while it leans, so to speak, on the causal notion—relates not so much to what happened but, rather, to *what should be done*. Accordingly, "responsible" here means "changeable," "treatable," "punishable." When used in this way, "responsible" usually means "bad" and carries with it the command to change—essentially to undo—the event so described. For example, in the case of the avalanche, the implicit command is to survey the conditions of snow and climate so as to prevent such occurrences in the future. Or, if we speak, as we do in medicine, of pipe smoking being *responsible* for a higher incidence of cancer of the tongue, we imply a command to avoid pipe smoking. This concept is fundamental to the notion of "treatment" in medicine and surgery. Finally, when our frame of reference is that of the law, and we say that "X is responsible for killing Y," our emphasis is on what should be done with X, rather than on what happened between X and Y. Accordingly, whenever legal considerations are at hand, the notion of responsibility is closely linked with that of punishability. In what follows, we shall consider the concept of "responsibility" in this light.

In everyday life there are two basic circumstances under which persons are not held "responsible" for their actions: (1) in the case of so-called "accidents," and (2) when the offender is a child. I shall not dwell on the question of free will directly in this connection, however important that question may be to our considerations. Instead, I shall try briefly to show the reasons for our usual judgments in these matters.

I think the chief reason why accidental damage to others is not punishable is because of the overwhelming role which identification with the offender plays in the current social

evaluation of this situation. As an example: A child plays on a street, runs after a ball, dashes in front of a car, and is struck down and killed by it. Since we can all imagine ourselves in the position of the driver, we feel that he should not be punished. It was, after all, as we say, "an accident." In this view of the matter, we focus on the intention of the driver, which did not include killing the child. We do not focus attention on the child or the parents as far as punishability is concerned. (The psychology of insurance compensation as a matter separate from "responsibility" enters here, but I shall not concern myself with this aspect of the problem.)

Another example: A soldier kills another man, designated as an "enemy." He, too, is not responsible. Here, again, in addition to other factors, the average observer's identification with the soldier is important. Most people can imagine themselves in this position and would not want to be punished for it.

A third example: A man kidnaps a child for ransom and later kills the child. In this case the offender is held "responsible" and is usually executed. The identification with the murderer here, too, plays an important part, but in this case by its reversal into the opposite. Here, the attitude of the public could be phrased thus: "I could not possibly commit such a terrible crime . . . the offender must be something less than human . . . it is society's duty to eliminate him from our midst." The evaluation of this situation is based largely on a complete repudiation of identification with the offender, which was shown to be so important in the previous instances. Moreover, the repudiation of the identification plays into the concept of "responsibility" and "punishability," by demanding the actual, physical elimination of the offender; this makes the continued effort of dis-identification with him unnecessary.¶

I trust it will be clear that I am not advocating either the punishment of the driver who kills by accident or the acquittal of the kidnaper, nor am I signifying my complete satisfaction with our present orientation regarding these matters. I am simply trying to call attention to psychological factors which seem important in determining our "common-sense" attitude toward accident, crime, and responsibility. An examination of such factors shows that our decisions concerning these situations have no relationship to the damage and suffering inflicted on various persons, but are dependent upon the degree and nature of our identification (or its repudiation) with the offender. This conclusion is, of course, by no means novel and is seen most clearly in connection with political actions: Whether a given act is considered heroic and virtuous or treasonable and criminal depends on a judgment of values by the observer. The complete at-oneness of the observer with the actor in the case of the heroic deed, and his complete dis-identification with him in the latter instance, are so obvious that it may escape one's notice that the entire judgment of the event, in such cases, rests on these processes.

Let us now consider the fact that children (legal "minors") are not considered (fully) "responsbile" for their actions. This notion derives, without much doubt, from the appreciation that there is some connection between the process of maturation—of becoming a fully developed human being—and such concepts as self-control, mastery over one's impulses, knowing right from wrong, and responsibility. Such is indeed the case, and, in accordance with this notion, the concept of responsibility is linked with such a hypothetical adult, possessing "knowledge," "character," "ego-strength," etc. Such a man is then said to have certain "rights" and "responsibilities." # Children and psychotics are

¶ For a discussion of various types of identification and related problems, see the recent contributions of Greenson (References 27 and 28), Jacobson,[32] and Reich.[43]

The word "right" here refers to the degree of freedom which the ego has vis-à-vis society, and the concept of "freedom" can be given meaning only in the context of "lack of restrictions." On the contrary, the term "responsibility" denotes that degree of "unfreedom" (constraint) which the ego experiences vis-à-vis the superego.

conceived of as having neither, or in any case are deprived of both.

In this complex question, I propose to focus on one particular issue, namely, on the notion that only persons endowed with relatively mature and intact egos can be considered responsible; and its corollary, that a demonstration or alleged absence of such ego functions is synonymous with "no responsibility." [16] I think this complementary relationship between ego development and responsibility accounts for much of our practical difficulty in applying psychiatric knowledge to legal change. For does it not follow that by a literal application of these concepts, we would place an additional legal burden on the shoulders of those most highly developed? Such individually specific liability to punishment would be a corollary to the relatively more highly developed egos and superegos which such persons possess, since, by definition, their very state of development would constitute the criteria for punishability. And, contrariwise, we would put a premium on the socially undesirable behavior of those who are (perhaps as yet) less highly developed.*

Let me illustrate what I mean. The concept of ego development is a relative one. It must be measured against something else, some other ego. Thus the child has an ego which, in many ways, is less developed than is the ego of his father or mother. However, an intelligent, well-educated teen-age child may well have a more highly developed ego than an illiterate "hillbilly." Yet the former cannot vote and cannot be sent to jail if he commits a crime. The latter is considered, for legal purposes, more "adult." It is clear that an enforcement of the psychiatric concepts currently proposed by many goes counter to some of our social and legal concepts of "equality" and "democracy." Instead of an impersonal set of "rights" and "responsibilities" which all men share, irrespective of creed, color, or education, a law embodying greater responsibility for those who are more highly developed (psychologically) would discriminate between persons as to punishability according to such a psychological criterion. Do the proponents of such changes also envision greater rewards for this elite class? † Further, who would determine the criteria according to which the so-called "ego impairment," or its absence, is ascertained?

Finally, it is important to note that from the point of view of scientific determinism ‡ —a philosophy to which psychoanalysis, as well as the social sciences, cannot help but adhere—the notion of "responsibility" is without any meaning whatsoever. Science aims at explaining how certain phenomena take place. It leaves no room for the possibilities of "choice" and "free will" and, consequently, in this frame of reference, the notion of responsibility does not arise. It is interesting to note, in this connection, that the deterministic philosophy regarding human conduct is always put forward as a plea for demonstrating that persons are not responsible for their offenses. A consistent application of such a philosophy to social life would also necessitate the assumption that people are not responsible for their so-called good, or socially useful, deeds either, and accordingly should not be rewarded for them. Such thoughts lead to rather bizarre conclusions when compared with our present cus-

* This latter consideration has some important roots in Jewish-Christian morality, as is shown, for example, by Christ's words on the cross, urging that those responsible for his crucifixion be forgiven for they did not know what they were doing. We touch here also on the larger problem of the connection between "knowledge" and "sin" ("guilt"), with which we cannot deal here in greater detail.

† This would be a logical corollary of greater "responsibility."

‡ It is a great error to think that the physical concept of "indeterminism" has undermined, and in some ways invalidated, the notion of determinism in the sense used in this paper, and in macroscopic physics in general. The so-called "principles of indeterminacy" relates to a thoroughgoing conceptual restructuring of the physical world in such a way that the specific identity of objects becomes unascertainable and meaningless; only under these conditions does the concept of indeterminancy become relevant.

toms. However, this line of inquiry promises interesting, and possibly important, implications for our understanding of group psychology. Perhaps a more deterministic philosophy of human conduct would bring about life without many of the complexities of rewards and punishment as we now know it. However, what man's experiences would be under such circumstances is something almost beyond our imagination.§

SOME ACTUAL RELATIONS BETWEEN PSYCHIATRY AND THE LAW

I now propose to examine the following two questions: 1. What is the function of the psychiatrist in the actual, current administration of the criminal law? 2. What is the psychological function of the law? These are two ambitious questions, but I will be satisfied for the present if I only indicate some tentative and partial answers to them.

In regard to the first question, I will differentiate between two aspects regarding the role of the psychiatrist in criminal cases: first, his official, consciously avowed function in the situation, and, second, his officially unrecognized, but psychologically "truer" role. Officially, the psychiatrist is supposed to be an "expert witness." This comes close to meaning that he is a scientist who in his role as an expert is supposed to enlighten the court. That is to say, he is a "teacher." Yet the very structure of the situation does not permit him really to function in this role, since the court is not a "student" whose wish is to learn psychiatry.‖ The court is supposed to *act* on a certain matter before it, and all the information which it receives is viewed in this light. In other words, the court is not a theoretician (as Freud regarded it in the passage quoted earlier) who is interested in "truth." This is not to assert its opposite, which is sometimes cynically maintained, namely, that the court is *not* interested in the truth. I would, rather, suggest that the structure of the situation in a court of law is such that the determination of the precise nature of events, in the sense of the scientist's intent on finding out how things happen, simply does not apply. This is rightly so, since how something has happened does not always tell us unequivocally how to proceed from there on, and this is exactly what the court must do. It follows, therefore, that anything a psychiatrist says in a court of law will be held either for or against the defendant. There is no place for neutrality there. To maintain that the psychiatrist should function as a "friend of the court" (Zilboorg [62]) seems to me a verbal nicety. This is advocated on the basis that the prosecution is assumed to favor guilt and conviction; the defense, innocence and acquittal, whereas the court itself is assumed to represent the interests of truth and justice. This formulation does not take a serious enough view of the actual, practical situation. While it is true that prosecution and defense usually function according to the foregoing motives, I maintain that it is an impractical ideal to believe that the court (particularly in the case of jury trials) can be an effective advocate of "truth and justice." The pursuit of truth is a complicated matter and is not facilitated by the pressure of having to make important, emotionally charged decisions within a reasonably short time. It cannot be maintained, therefore, that the psychiatrist

§ Cf. Samuel Butler's "Erewhon" [11] for an ethical orientation to illness and crime which is just the opposite of our own. However, it seems to me that the bizarre "culture" portrayed in this fantasy is actually much closer to accustomed modes of thinking—since it is essentially nothing but its opposite—than would an ethical system based on a deterministic philosophy of behavior.

‖ Glover,[25] p. 109. "The law, in short, is not primarily interested in psychopathology; it rarely goes further than to characterize certain end products of psychic tension (behaviour) as socially undesirable or reprehensible, and to charge and sentence the offender accordingly. Even when, as in the case of the McNaghten Rules, the law established the relative immunity from punishment of offenders suffering from some forms of mental disorder, its concern is less with the actual state of mind of the individual than with the lack of social responsibility ensuing there from."

can help the court to arrive at the "truth" of the situation.¶

What is left is "justice," which I believe should be considered a matter of *social convention.*# The psychiatrist in his role as a scientist has nothing to contribute to this, except to emphasize that justice is man-made and never derived from any "higher authority." Science does not tell us what is just or unjust—it only tells us that we can never be *sure* of either. If the psychiatrist does testify in a court of law, whatever his role might be called, he testifies as a citizen who has every right to be interested in the law.

Lest my position be misinterpreted as a nihilistic one, I want to add that, while for the reasons stated I do not believe that psychiatric theory as such has any relevance in a courtroom, I do think that insofar as changes in criminal law may be desirable, psychiatric considerations may be validly employed on the level of legislation to be drafted. To be valuable on this level, however, I think much more need be done than to show how present judicial methods are ineffective in achieving their alleged aims. While that may be true, it must be remembered that not all innovations are improvements.[39] To mention but a single relevant example, we might contemplate whether the sexual enlightenment of children, so much wished for in the early days of psychoanalysis, has made the ego development of children any "better" than it was in the past.

Let us turn to the problem of the psychological function of the law. Several such functions have been described and emphasized by previous authors.* There is general agreement that the law, as a social phenomenon, represents an equivalent of the individual conscience, or superego. The three chief aims of the law—punishment, retaliation, and correction—are subsumed under this concept.

Such an interpretation of the psychology of the law is, in my opinion, valid but too narrow. It does not take into account many other psychological functions of the legal framework of rules of conduct; it may also err by ascribing the so-called irrational aspects of the law predominantly to man's sadistic impulses and to its derivatives, the primitive, punitive superego.†

¶ The notion of a "scientifically" impartial and just court of law seems to me in large measure a sentimental belief in an unconscious wish, not radically different from that of a "heavenly court," where "divine justice" reigns. If science is to make any contributions to this aspect of human life, it will have to do so, in my opinion, first by making an explicit and candid analysis of the psychological and social structure of the law.[45] Consider, in this connection, Auger's [4] classification of man's search for "understanding" into three categories according to the three basic questions of "Who?" "Why?" and How?" In a brilliant analysis, Auger sketches the evolution of "understanding" through these three stages; that is, from anthropomorphic cosmology, through a "natural law" that is akin to human "will," to the present era of natural science, which aims only at "order." If we apply these notions to psychiatry and the law, it becomes immediately apparent that the latter (particularly criminal law) is interested in the question of "Who . . . (did such and such)?" Psychiatry and psychoanalysis, insofar as they deal with "motives" and "drives," are oriented to the second question, as if they were to ask "Why . . . (did X do such and such)?" Modern physics (and, hopefully, the psychology of object relationships, rather than drives) focuses on the question of "How . . . (do certain events relate to each other)?"

Aristotle believed that slaves were inferior beings who were justly subdued and deprived of their liberty (cited by Einstein [17]).

* References 3, 58, 60, and 62.

† The idea that crime is due to "hatred" is an extremely popular and convenient oversimplification. To substitute for this the psychoanalytic notion of "aggression" actually does not take us very far beyond the ancient moral view of this matter which attributed crime to man's "evil nature." Cohen's "Human Behaviour in the Concentration Camp" [12] is an important document in this connection, for he shows clearly that the notion of aggression cannot be used as the chief explanation for the action of the German people in that situation. "It was not hate, but the moral current in Nazi Germany that made the extermination of the 'inferior' races possible" (p. 241). "Moral current" here refers not to ethics in the narrow sense but to the sociology and psychology of a national group.

I wish to call attention to some other psychological functions of the law, which in individual psychology are more closely analogous to ego functions than to the functions of the superego. I have in mind all those functions of the law which provide a relatively fixed, stable framework of rules by which man has to conduct himself. Traditionally, these have been looked upon as "restrictions," much as the parental attitude to the child was regarded as various ways of thwarting the child's instinctual needs. While I do not want to imply that this point of view is "wrong," I do not want to stress that it represents, so to speak, only half of the story.

In other words, while certain parental attitudes may be regarded as instinctual deprivations, they also have elements of "structuring" an otherwise chaotic world. All this is well known. However, it is often thought that there is some hypothetical ideal toward which one should strive that would include a minimum of instinctual deprivation and a maximum of "security in order." I believe this is based on a fundamental misunderstanding which attaches a positive evaluation to instinctual satisfaction and a negative one to the "restrictive" connotation of "order." Actually, the two notions refer to one and the same process.

It is our belief today that the child needs a well-ordered environment, in the sense of fairly rigid and consistent rules regarding what he can and cannot do. The emphasis here is decidedly on rigidity and consistency. Why is this "good"? It is good because it simplifies matters, and a relatively immature ego can thus acquire techniques of adapting to various situations in small doses. As in learning, it is a matter of tackling only as much as one can handle at the time.

Our concept of a "good and just" law is analogous in many ways. We do not consider the law to be just if it does not apply equally to everyone (i. e., to all the children), or if it applies in different ways under different circumstances (i. e., depending upon how the parent feels at the time). In all these ways, we expect and need the law to be rigid and inflexible.

It is interesting to compare this with our concept of the nature of science.‡ Without entering into the full complexities of this matter, I want only to emphasize that the psychological function of science is complementary to that of law: While the law is supposed to be rigidly fixed and unchanging, at least over appreciable periods of time, science is expected to be ever tentative. I believe that it is in this specific psychological function that the most important difference between science and law (and religion) rests. In other words, it is *not the content* of the explanations—where the difference has for so long been sought—which enables us to distinguish science from religion (or science from paranoia) but the way in which man relates himself to these "symbols."

The psychological need for science and for laws of all sorts was described with great lucidity by Susanne Langer in connection with her studies of the nature of symbolization. She wrote as follows:

> The story of man's martyrdom is a sequel to the story of his intelligence, his power of symbolical envisagement. For good or evil, man has this power of envisagement, which puts on him a burden that purely alert, realistic creatures do not bear—the *burden of understanding*. He lives not only in a place, but in space; not only at a time, but in History. So he must conceive a world and a law of the world, a pattern of life, and a way of meeting death. All these things he knows, and he has to make some adaptation to their reality. Now, he can adapt himself somehow to anything his imagination can cope with; but he cannot deal with Chaos.§

Closely related to the notion that the function of the law is to bring order into the realm of human conduct is its role of protecting people from a feeling of (unconscious, unexplained) guilt. I shall assume for the present that this feeling arises in everyone and that various meaures are taken by man

‡ References 39, 48, and 59.

§ Reference 37, p. 233.

to avoid it.‖ How does the law fulfill such a function? Perhaps, with a slight oversimplification, one may paraphrase the answer as follows: "I am a God-fearing and law-abiding citizen . . . therefore I need feel no guilt. *If* I was guilty, I would be prosecuted and punished." This aspect of the law (secular as well as religious) brings to the fore its nonrestrictive, ego-defensive functions.

One direct application of the foregoing concepts to medicolegal operations may now be mentioned. When the psychiatrist is called into court to testify whether a man is "insane" or not, I believe what he is really asked to do is something like this: The court has a set of rules by which it orders its behavior. It (i. e., the jury, judge, etc.) does not feel guilt if it convicts a man according to its set rules. However, once it is told that it must not punish a man who is "insane," it must have assurance that the man is "sane." Accordingly, the "responsibility" (and the need to dispose of the possible feelings of guilt) is shifted to the psychiatrist. Thus, in effect, the psychiatrist is asked by the court to give it *assurance* that it can proceed with punishment *without feeling guilt*.¶ I think it may be no exaggeration to assert that in many cases this may be the only real function of the psychiatrist in the legal handling of a criminal case. Perhaps this explains why the psychiatrist is pushed toward the role of almost always pleading for the defense. Such considerations may be helpful also in showing how the powerful urge to avoid feeling guilty may drive man to erect complex social organizations in which these motives and feelings are hidden and discharged in a disguised and diluted form. The foregoing analysis of the role of the psychiatrist in criminal law may account in large part for the logical absurdities to which lawyers and medical men are driven by these unconscious forces.#

Acceptance of the foregoing considerations leads to some unavoidable inferences. First, we would have to conclude that the suggestion to make the law more "humane" by making it more "flexible"—for example, by various assessments of ego development—would constitute not simply a practical revision of the law in the light of so-called scientific facts, but would amount to a fundamental change in concepts. In other words, insofar as the human need for "externally" ordered conduct persists, altering the law so that it becomes more flexible is tantamount to removing such principles from the confines of what constitutes "law." If we agree on the complementarity of the psychological functions of the law and of science, then we cannot have a "scientific law," in

‖ The possible sources of this feeling need not concern us in this essay. That feelings and ideas of "sin," "guilt," and "worthlessness" are ubiquitous, if indeed not universal, requires no documentation (consider, for example, the notions of "original sin," the "burden of understanding," and the intimate connections between object loss, mourning, and guilt). Accordingly, irrespective of the possibly diverse sources and "meanings" of guilt-feelings, I wish to propose that one of the important functions of the "law" (secular, as well as religious) is to provide a social framework in which people need not feel guilty (Kafka [36]).

¶ The legal orientation toward the sentencing of persons who are physically ill or aged, even if their "guilt" and "responsibility" are assumed to be proved, shows clearly how strongly the court is governed by its desire to avoid feelings of guilt. For an illustration of this thesis, consider the following material taken from a recent issue of *Time* [52]:

> "Died. Israel Amter, 73, one of the founders (in 1919) of the Communist Party in the U. S.; of Parkinson's disease; in Manhattan. A pianist and composer by profession, Amter was a tireless leader of demonstrations of the unemployed, frequently his party's candidate (unsuccessful) for U. S. senator, governor, and mayor of New York. He was included in the 1951 indictment (for conspiring to overthrow the government by force) which sent 15 top Communists to jail, but *was not brought to trial because of ill health.*" (Italics mine)

It is important to note, in this connection, that a diametrically opposite type of social action takes place once those in power feel free of guilt toward the "offenders." Thus, Cohen described how it was possible that the Nazis in charge of concentration camps killed precisely those people toward whom a civilized court shows the most guilt, namely, children, old people, and the sick.[12]

which each case is judged on its own "merits"; in actual practice, this is impossible, and simply leads to a perversion of both law and science.*

These thoughts lead to still another inference, this one relating to the changing types of "morality" as man matures. As we know from individual psychology, most reasonably well-developed persons do not need external restrictions to enforce their law-abiding behavior. The more highly developed man is, the more "ordered" his conceptual universe becomes and the less dependent he becomes on external rules (law, religion, myth, superstition, etc.) to provide the necessary orientations for daily living. Perhaps one may consider, along this line, science as a "higher form" of morality (in the sense of its being a more complex development) than either religion or secular law. And, indeed, we know from everyday life that scientists oftener place themselves "above the law" of the land than do other people. However, these are considerations which lead beyond our present topic and are best left for further contemplation.

SUMMARY AND CONCLUSIONS

There is a widespread interest today in applying scientific knowledge to problems of everyday life. In the physical sciences this tendency gave rise to our present technology. The influence of the psychological sciences on society as a whole has been less impressive. This corresponds, in a general way, to the fact that we know much less about the laws governing human relationships than about the laws of inanimate nature. At the same time, the impact of psychoanalysis on Western civilization must not be underestimated. It has influenced, and changed, the education of children, literature, the world of entertainment, and the pattern of advertising, to mention but a few examples. The influence of psychoanalysis on the administration of the law has been, so far, relatively slight. Some psychiatrists and psychoanalysts believe that the science of human behavior could be applied to legal matters—particularly to the handling of criminals—in such a way as to make these procedures more humane, more rational, and more "just." Others, notably George Orwell, in his famous Utopia of horror, "Nineteen Eighty-Four,"[40] point out how scientific knowledge about human feelings and behavior might also lead to results which seem undesirable and dehumanized to most of us.

Indeed, we have no reason to expect otherwise. The physical scientists have had to meet this problem long before it has become much of an issue for the psychologist and psychiatrist.[6] It is hardly necessary to belabor this point further, living as we now do in an era of anxiety about whether nuclear energy will be used for peace or for war.

The psychologically impossible situation into which the psychiatrist enters when he compounds his role of scientific expert with that of a so-called "responsible citizen" is brought out well in Davidson's words [15]:

> "The mechanism of the impulse to murder (in juvenile murderers) has been analyzed by Bromberg and by Alexander and Healy. The psychodynamic explanations are interesting and well expounded in the psychiatric literature. However, these explanations are of little practical value to the court which has the "responsibility" for 'disposing of' these young offenders. From a practical viewpoint, the significant factor is that juvenile murderers, whether psychopaths, defectives, or prepsychotics, are serious threats to the community because the dynamic mechanism which led to one murder is not likely to be changed by treatment or imprisonment. Making a recommendation to the court, thus imposes on the psychiatrist a peculiar responsibility, one which highlights the tragic dilemma of the immediate interests of his *patient* compared with the interest of society as a whole. No psychiatrist wants to be *responsible* for a death sentence or for a sentence of life imprisonment, particularly when the defendant is a sick child. Yet he cannot, in clear *conscience* be the instrument for allowing such a dangerous child to be returned to the community" (pp. 157-158).

Note the repeated emphasis on responsibility and the underlying, almost explicit, motive of not wanting to be guilty which governs the psychiatrist in this dilemma.

* See in this connection Szilard's [50] excellent satire on how the use of our knowledge of motivational psychology may lead to the disenfranchisement of people.

The purpose of this essay is to examine the relationship between psychiatry and the law. Its point of departure is the thesis that the *application* of scientific knowledge about human feelings, motivation, and behavior is not *necessarily* "beneficial" for the individual or for mankind. A survey of the psychiatric literature on this subject shows that current opinion leans heavily toward viewing the influence of psychiatry on criminal law as something inherently desirable. According to this view, psychiatric knowledge is bound to humanize and to "improve" the legal handling of offenders. Whether this is so or not is not easily ascertained. In any case, I have attempted to show that the basis on which judicial changes are advocated by many psychiatrists rests on concepts which are either ill-defined or borrowed from other areas of thought (e. g., medicine) and cannot be applied directly to the social problem of antisocial actions. The two most important concepts which we encounter in this connection are "mental disease" and "responsibility." A critical examination of these concepts is a prerequisite for a clarification of the relationship between psychiatry and law.

The notion of "mental disease" is patterned after the concept of "physical disease" and is borrowed from medicine. This is not surprising in view of the fact that modern psychiatry and psychoanalysis have originated from medicine. Yet the source of origin is not the sole determinant of the nature of a body of thought which, like a living organism, grows and in its process of development may become significantly different from its parent. Indeed, so it has happened with psychiatry. For we find that we cannot define, or give meaning to, the notions of "mental health and disease" in the same way as we can define physical health and sickness. It is suggested that whenever we propose a definition of mental health, we simply state our preference for a certain type of cultural, social, and ethical order. And, conversely, we designate deviations from such an explicitly stated, or more often hidden, model as "mental disease."

Examination of the problem of "responsibility" leads to the conclusion that we must distinguish sharply between this term denoting a question regarding "what happened?" (causal connection) and another question regarding "what should be done?" In the first instance, the questioner aims at understanding a sequence of events; in the second, his aim is to change, to modify, to act. Or, from still another point of view, the first question is oriented to the past, whereas the second is oriented to the future.

The foregoing two orientations and questions complement each other. We are familiar with this complementarity from a number of sources. In physics we speak of "pure science" and "technology"; they reflect respective primary orientations toward explanation (understanding) and action. In psychoanalysis, we encounter the same phenomenon in the complementarity of theory and treatment. These considerations have particular relevance to the problem of the relationship between psychiatry and law. For psychiatry, as a science, is focused on *understanding*. The law, on the other hand, is a social institution so constituted that it must focus on *action*. It is analogous to technology rather than to theoretical science.

The term "responsibility" used in a legal context is therefore very close to meaning "punishability." When destructive events occur in which people play a role, there is a search for the "responsible party." There are two basic conditions when, in spite of damaging events, no one is held to be "responsible": in the case of so-called accidents and when the offender is a child. These judgments characterize our present orientation to such events. As we know, in bygone times and in other cultures, persons have been held "responsible" and punished both for what we now call accidents and when the offender was a child. Accordingly, we are justified in asking why it is that in our present society we judge these events as we do. A discussion of this subject is presented, and it is suggested that our present judgments regarding these matters are based primarily

on an (unconscious) *identification with the offender*. This leads to two basic outcomes. 1. In situations in which the observer's identification with the offender is unblocked and acknowledged (although this acknowledgment, too, may be, and usually is, unconscious), the judgment reached is that the offender is not responsible and should therefore not be punished. Examples of such situations may be seen in the case of automobile accidents or when a person commits destructive acts under socially acknowledged pressure of authority (e. g., warfare). Similarly, children are not regarded as autonomous persons, do not have the "rights" of adults, and are not held responsible for crimes. 2. The alternative outcome of the process of identification with the offender is that of complete, often aggressive repudiation (on an unconscious level) of the identification. Examples of this phenomenon are seen in the so-called crimes of violence, particularly against children (e. g., kidnapping or killing a child), and in cases of sexual offenses. In these situations the offender is judged to be "responsible" and meting out serious punishment is felt to be "just."

Introduction of the psychiatric notion of "mental disease" as a basis for relative lack of "responsibility" presents us with an interesting and important conceptual and philosophical problem. This is due to the fact that an examination of the principles underlying the foregoing connection between "mental disease" and "responsibility" leads to the conclusion that in actual legal practice increased psychological maturity would be penalized by greater "punishability"; and, conversely, offenders judged mentally ill would be afforded protection from punishment without, however, loss of their customary civil rights. If this line of thought is pushed further, it leads easily to a point of view according to which the "psychologically mature" would be an elite class and those less developed would be relatively disenfranchised. These and some other implications of the application of psychoanalytic concepts to the law are discussed. It is evident that a thoughtful deliberation of such issues would serve as a necessary safeguard against untoward results which might follow from a hasty and overly enthusiastic application of psychoanalytic principles to legal matters, however passionately these may be advocated in the name of humanism.

In conclusion, some actual interrelations in the everyday functioning of psychiatry and of the law are considered. It is suggested that probably the chief unconscious function of the psychiatrist in a court of law consists in taking unto himself a large measure of the guilt which the court (judge, jury, prosecution) would feel in sentencing (i. e., damaging) a man. Once there develops a widespread recognition that persons who are "mentally ill" should not be punished, it becomes necessary for the court to be assured—whenever there is doubt on this score—that it is "playing the game" according to the rules. It is for this purpose that the services of the psychiatrist are enlisted. Accordingly, the psychiatrist is asked to render an opinion as to whether the defendant is sane or not—a judgment which, in fact, has no meaning other than whether he may be punished with a clear conscience. It would seem that when psychiatrists testify—irrespective of the nature of their testimony—they, in fact, participate in this complex unconscious "game" of dissipating and projecting feelings of guilt unto others.

These considerations also raise the question of the psychological functions of the law for the human psyche. No detailed study of this important problem is attempted in this essay. According to the traditional psychoanalytic view, the law is considered to be a social analogue of the individual superego, and is often viewed specifically as a form of counteraggression pitted against the criminal's aggression. This view is misleading because it places exclusive emphasis on what is probably but one—however significant—facet of this problem. It is suggested that another important function of the law lies in furnishing a social structure by which man can order his relationships with others in such a way that he need not experience an unremitting feeling of (unconscious, unex-

plained) guilt. By matching his overt behavior against the explicit rules of conduct embodied in the law, he can remind himself that he is "innocent." Clearly, this function of the law requires it to be relatively rigid and fixed, in contrast to science, which must be forever tentative and hypothetical. It thus places a conceptual paradox in the way of realizing the often-proclaimed ideal of a humanistic law before which each man would be judged on his own "merit."

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News and Comment

SOCIETY NEWS

North Pacific Society of Neurology and Psychiatry.—The annual meeting of the North Pacific Society of Neurology and Psychiatry will be held at the Gearhart Hotel, Gearhart, Ore., on April 12, 13, and 14. Dr. Wendell Hutchens, president, Portland, will preside. The guest speaker will be Dr. Theodore Rasmussen, Professor of Neuro-Surgery at the Montreal Neurological Institute. For further information, contact Dr. John Evans, secretary, 919 Taylor St. Bldg., Portland, Ore.

Scientific Exhibits

PHARMACEUTICAL TREATMENT OF PATIENTS WITH SENILE BRAIN CHANGES

LEOPOLD HOFSTATTER, M.D.; ABEL OSSORIO, Ph.D.; BILLIE MANDL, Ph.D.;
LOUIS H. KOHLER, M.D.; ANTHONY K. BUSCH, M.D.,
AND
ANN HYMAN, M.D.
St. Louis

Shown as a scientific exhibit of the Section on Nervous and Mental Diseases at the 104th Annual Meeting of the American Medical Association, Atlantic City, June 6-10, 1955.

Rationale of Therapeutic Attempts

Mild and prolonged vaso-dilatation
Stepped up cerebral blood flow etc.
Dihydrogenated ergot alkaloid compound Hydergine
Nicotinic acid tartrate (Roniacol)

Cerebral cortical stimulation
Increase in nerve impulse transmission
Pentylene-tetrazol (Metrazol)

Method of Study

SELECTION OF PATIENTS:

Thirty five out of 57 patients with signs of advanced senescence were amenable to objective study.

GROUPING OF PATIENTS:

Three groups of patients (A, B, C,) were formed and equated for age, duration of illness and education *(see Table)*

ADMINISTRATION OF SELECTED DRUGS:

Group A (11 patients) received 2 dummy tablets t.i.d., p.c.
(Control)

Group B (13 patients) received 2 Hydergine tablets (0.5 mg) t.i.d., p.c.
(Experimental)

Group C (11 patients) received 2 Roniacol tablets 50 mgm
(Experimental)
plus
1 teaspoonful Elixir Metrazol 100 mgm t.i.d., p.c.

A "blind" procedure was followed in the administration of drugs with patients unidentified to ward and medical personnel as to type of medication.

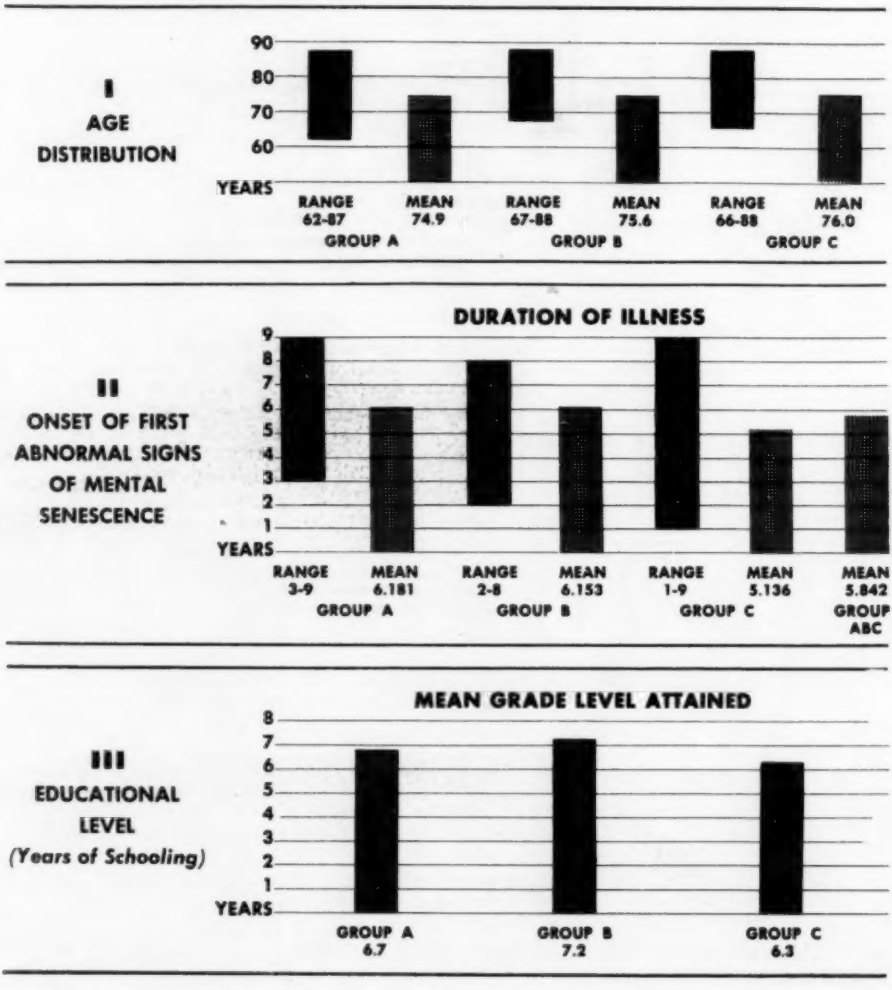
EVALUATION OF MENTAL AND BEHAVIOR CHANGES:

Objective rating of behavior and psychological tests (*see Table*) were obtained one week prior to therapy and in the sixth week of drug administration.

A "blind" procedure was followed in all psychological measurements with patients unidentified to raters as to type of medication.

This is a reproduction of the chart used in the exhibit. The author indicates that "Nicotinic acid tartrate (Rouiacol)" should be "the tartaric acid salt of the alcohol corresponding to nicotinic acid."

Composition of Study Groups



Test Battery and Objective Rating Measures Developed to Distinguish Changes in Status

MEASURES OF EFFICIENCY OF INTELLECTUAL FUNCTIONING

Memory
Knox Tube Test, Object Memory Test, Wechsler Memory Scale (Digits), Rating Scales

Abstract Ability (Reasoning)
Goldstein-Scheerer Cube Test

Alertness-Learning Ability
Reaction-Time Discrimination Learning Test

Mental Control
Wechsler Memory Scale Subtest

Associational Fluency
Word-Speed Test

Flexibility of Thinking
Geometric Figures Test

Attention & Concentration
Behavior Rating Scales

Orientation
Behavior Rating Scales

MEASURES OF MOTOR COORDINATION AND ENERGY OUTPUT

Energy Output
Tapping Test, Reaction Time Test, Ratings of general activity level

Motor Coordination and Motivation
Seguin Form Board

MEASURES OF PHYSICAL STATUS

Appetite
Behavior Rating Scales

Feelings of Physical Well-being
Rating Scales

MEASURES OF EMOTIONAL STATUS

Feeling Tone or Mood
Senile Apperception Test, Sentence Completion Test, Behavior Ratings

Emotional Reactivity
Behavior Rating Scales

Temper Control
Behavior Rating Scales

MEASURES OF GENERAL SOCIAL INTEREST

Characteristic Attitudes Towards Others
Behavior Rating Scales

Sociability
Behavior Rating Scales

Cooperation (Conformity)
Behavior Rating Scales

Participation in Recreational Activity
Behavior Rating Scales

Readiness to Communicate
Behavior Rating Scales

Attentiveness to Environment
Behavior Rating Scales

RESULTS

OBJECTIVE INDICATORS OF IMPROVEMENT

A. Changes towards improvement in Group B (Hydergine Treatment) In Comparison with Group A (Control Group receiving no drug)

	FAVORABLE TRENDS*	FAVORABLE CHANGES**
1 Efficiency of Intellectual Functioning		
1. Memory	2	1
2. Mental Control		
3. Attention	1	
4. Abstract Thinking	1	1
5. Mental Fuency	1	
2 Motor Coordination & Energy Output		
1. Activity Level		
3 Physical Status		
1. Appetite	1	1
4 General Social Interest		
1. Characteristic Attitudes Towards Others	1	
2. Cooperation (Conformity)		2
3. Sociability	1	
4. Readiness to Communicate	1	
5. Participation in Activity	1	

B. Changes towards improvement in Group C (Nicotinic Acid Tartrate-Metrazol Treatment) In Comparison with Group A

	FAVORABLE TRENDS*	FAVORABLE CHANGES**
1 Efficiency of Intellectual Functioning		
1. Memory		1
2. Mental Control	1	
3. Flexibility of Thinking	1	
4. Associational Fluency	1	
2 Motor Coordination & Energy Output		
3 Physical Status		
1. Activity Level	1	
4 General Social Interest		
1. Sociability	1	
2. Readiness to Communicate	1	
3. Participation in Recreational	1	

*Change in a favorable direction approaching, but not attaining, statistical significance.
**Statistically significant changes in a favorable direction (P Values .025 to .001).
Evaluation of statistical significance by non-parametric and t-tests of significance.

ADDITIONAL FINDINGS

1 Group A (Control Group) showed insignificant changes in status in both positive and negative direction.
2 Groups B and C showed no statistically significant change in a negative direction in any measure.
3 In all groups, patients with pronounced paranoid symptoms tended to be less amenable to testing, treatment, and improvement.

COMPARISON

OF PSYCHOLOGICAL TEST FINDINGS AND BEHAVIOR RATINGS OF EACH GROUP BEFORE AND AFTER TREATMENT

Improvement (+) and decrement (−)

	CONTROL GROUP A PLACEBO		HYDERGINE GROUP B		RONIACOL-METRAZOL GROUP C	
	Trends approach-Signif.	Statist. Signif.	Trends approach-Signif.	Statist. Signif.	Trends approach-Signif.	Statist. Signif.
1. *Efficiency of Intellectual Functioning*						
1. Memory	+				−	
2. Mental Control	−				−	
3. Attention			+			
4. Abstract Thinking						
5. Alertness–learning ability	+			+		+
2. *Motor Cordination & Energy Output*						
1. Activity Level	+			+		+
3. *Physical Status*						
1. Appetite	−		+			
2. Physical well being	+		+			
4. *General Social Interest*						
1. Characteristic Attitudes towards others	−					
2. Cooperation (conformity)	−		+		+	
3. Sociability	−		+			
4. Readiness to communicate					+	
5. Participation in recreational activity					−	
5. *Emotional Status*						
1. Emotional responsiveness	−		+			
2. Mood	−		+			

✚Evaluation of significant changes (P Values .025 to .001) by non-parametric and t-tests of significance.

PRELIMINARY CONCLUSIONS

I. UNDER PROPER GENERAL MANAGEMENT, THE INTRODUCTION OF DRUG THERAPY MAY RESULT IN SIGNIFICANT IMPROVEMENT IN THE STATUS OF PATIENTS WITH ADVANCED SIGNS OF SENESCENCE.

II. PSYCHOLOGICAL TESTS AND OTHER OBJECTIVE MEASURES USED IN THIS STUDY INDICATE IMPROVEMENT, UNDER DRUG THERAPY, IN SOME ASPECTS OF INTELLECTUAL FUNCTIONING, PHYSICAL STATUS, AND SOCIAL PARTICIPATION.

III. IN THE GROUPS STUDIED, A GREATER NUMBER OF MEASURES SHOWING FAVORABLE CHANGE IS FOUND IN THE GROUP RECEIVING HYDERGINE.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Erich Lindemann, M.D., *President, Presiding*

Regular Meeting, May 19, 1955

Central Effects of Strychnine on Spinal Afferent Fibers. Drs. J. Y. Lettvin, W. S. McCulloch, W. Pitts, and P. D. Wall.

First, we wished to test some of the many theories on the action of strychnine by determining its effect on the excitability of motor horn cells and of terminal aborizations of spinal afferent fibers. A stimulating microelectrode was placed in the ventral horn of a lumbar spinal segment in anesthetized curarized cats. Recordings were taken of the antidromic volley running in the Group 1 fibers in the dorsal root and of the orthodromic volley running in the ventral root of the segment. After intravenous strychnine no change was found in the threshold of the motor horn cells. The terminal aborizations of the afferent fibers showed an increase in threshold. As further evidence for action on primary afferent fibers, it was shown that strychnine affects all components of the electronic dosal root potentials. The earliest components, believed to be due only to the passage of impulses in the primary afferent fibers, were affected. In these respects, strychnine imitates post-tetanic potentiation of reflexes, which is due to hyperpolarization of the stimulated fibers. No evidence could be found of a decrease of threshold of the motor horn cells after strychnine.

Second, the nuclei of Goll and Burdach were locally strychninized, and recordings were taken from the dorsal columns and lumbar dorsal roots. It was found that synchronized antidromic volleys generated in the strychninized areas ran down the dorsal columns and out of the dorsal roots. The caveats of strychnine neurography were discussed. Five examples are known of strychnine failing to fire off central nerve cells; one example of transsynaptic conduction of strychnine "spikes" has been shown in the sympathetic nervous system, and above we show antidromic spike propagation in the dorsal columns. So long as such exceptions are considered, strychnine neuronography remains a useful tool as an indicator of organization and as a guide for definitive anatomical studies.

DISCUSSION

Dr. Paul I. Yakovlev: I should like to ask Dr. Wall what is his understanding of the action of strychnine on the neuron. How does it change the phenomenon of excitability and conduction?

Dr. P. D. Wall: We are not suggesting that we have shown the complete action of strychnine. There are two general phenomena one sees in an animal under the influence of strychnine: hyperexcitability, and synchronization, which may become organized into a tetanus. Strychnine may affect the metabolism so as to increase the polarization across the membrane and produce the hyperexcitability by an increase in the height of the action potential. This is not an adequate explanation of rhythmicity. The question of how a drug increases polarization is beyond the scope of our work.

Dr. J. Y. Lettvin: If you paint a region with strychnine, you have all the cells fire off in synchrony. One would expect that they do so because of a common signal to them. This suggests that strychnine activity is initiated in the fibers of termination. The reason the Goll and Burdach experiment was important was that this region was the only one where we could be sure no efferent fibers could corrupt the picture. We would like to know whether there are other regions where input and output are not mixed and which are accessible.

Dr. Jerome K. Merlis: There are many reports in the literature indicating that after strychnine, cells may show a depression to evoke responses at the same time as they are firing strychnine spikes. Do you have any information about the action of strychnine on the synaptic inhibitory arc?

Dr. P. D. Wall: Although the work of Eccles shows a decrease of inhibition with strychnine, our own work and that of others indicates that some types of inhibition may be increased. This effect and the possible hyperpolarization of elements make it difficult to predict what will

happen to the threshold of an evoked response. Fibers are more difficult to fire off when hyperpolarized. Once you can get them to fire off, the result is greater.

DR. PAUL I. YAKOVLEV: There is hardly a single fiber system, however compact, that is 100% polarized in one direction. It is a matter of density.

DR. P. D. WALL: The evidence for antidromic volleys from the nucleus of Goll was from dorsal roots. I do not know of any evidence for descending fibers which pass out in the dorsal roots.

DR. PAUL I. YAKOVLEV: In lower forms descending fibers in the dorsal columns are well known. Even in cats and monkeys one would expect some descending fibers from the reticular formation.

DR. P. D. WALL: But the antidromic strychnine spikes which we have shown here not only descend the dorsal columns but also run out of the dorsal roots, whereas the descending fibers which you mention do not pass out from the cord and run in the dorsal roots.

Frontomedian Leucotomy for Pain: Psychiatric Observations on a Patient Before and After Surgery. DR. JOHN C. NEMIAH and DR. JAMES C. WHITE.

A 40-year-old paraplegic man underwent a Grantham frontomedian leucotomy for intractable pain in his legs. Preoperatively he was agitated and depressed, complaining of pain. In psychiatric interviews he repeatedly responded to questions about his concern over the effect of the injury on his life situation with denial of such concern and complaints of pain. Accordingly, he was interviewed before and after leucotomy and questioned about the effect of paraplegia on his life situation. The interviews were tape-recorded and analyzed for the number of pain and situational responses. There was a marked shift from pain responses before operation to situational responses after operation (significant at the 0.001 level of probability as determined by the χ^2 test). Postoperatively he showed no anxiety nor depression; when questioned, he admitted pain, but there was no spontaneous complaint of it. It is postulated that leucotomy physiologically reduced the patient's ability to experience anxiety; as a result, the patient could face direcly problems around his life situation which had previously created sufficient anxiety to cause a displacement to his pain of conflicts centered around being paraplegic, thereby emotionally increasing his suffering from pain.

DISCUSSION

DR. ERICH LINDEMANN: This study is notable because it gives us increased rigor in evaluation on both sides. We are more precise concerning the lesion of the brain, and we are more precise in appraising the mental status. The use of content analysis of interviews is not as common as it could be. This makes it possible to relate neurophysiological data to some more dynamic aspects. This, again, is a badly needed exercise.

DR. PAUL I. YAKOVLEV: This paper is a most interesting study of a patient with severe physical disability. According to the preoperative content analysis, there was a great incidence of complaint of pain and distress—anxiety, an accompaniment of distress—and less concern about the meaning of paraplegia. After the operation the concern about the complaint of pain receded into the background, whereas the concern about the meaning of paraplegia connected with livelihood came more into the foreground. This is somewhat different from the general impression one gets from reports about surgery for pain.

DR. JOHN C. NEMIAH: Before surgery the patient showed a great deal of affect-anxiety and depression. After surgery, there was little, and the patient was apathetic. Before surgery the anxiety and concern were about his pain; when he was asked a stimulus question about his life situation resulting from paraplegia, he would appear upset but deny concern, and complain of pain. After surgery, in response to the same stimulus question, he would talk about his life situation but without affect. In other words, following surgery, there was a diminution of affect, as has been described, and a change in the content of his responses to the questions.

DR. PAUL I. YAKOVLEV: This presents a rather new point.

DR. JOHN C. NEMIAH: I think we have to distinguish between the concern (the affect) and the content. His concern was not really enhanced after operation; on the contrary, it appeared diminished. With the diminished affect, the content changed in that he talked about the effect on his life of his paraplegia, and not about pain. It was on this basis that we inferred that preoperatively the concern over being paraplegic was displaced to the pain, thus increasing his suffering from it. His talking about the effect of paraplegia postoperatively was not concern.

DR. PAUL B. JOSSMANN: The exactness of this new neurosurgical procedure represents a milestone in the history of psychosurgery. The accuracy in placing the lesion and the determination of its size are a safeguard against the previously uncontrollable damaging side-effects. Dr. White's observations with repeated operations reopens the discussion of the working mechanism of lobotomies and seems to emphasize the quantitative factor involved. The reduction of anxiety is an acceptable working hypothesis. Still the question remains as to what causes the reduction of anxiety physiologically.

DR. JAMES C. WHITE: I am sure that the amount of frontal lobe removed is more important than the area. We have experimented with different methods. In a nonpsychotic patient with pain, after the old Freeman-Watts procedure of transecting the entire bundle of white matter on the plane of the coronal suture, you get a patient totally devoid of affect whose social behavior is difficult, to say the least. If you transect only the two lower quadrants, you get a patient who is apathetic but without the bad behavior problems. Then, if you use Scarff's method of complete unilateral leucotomy, you get good relief of pain, provided the patient does not live for more than six months. Such a patient is not a behavior problem, but is not as devoid of psychological abnormalities as Scarff has claimed. Dr. Cobb and Dr. Bonner found some degree of apathy, diminution in amount of speech, and increase in amount of prodding they had to have to keep these patients talking. The procedure described here is something like the bilateral medial lobotomy described by Dr. Solomon. These patients do better than others with the Freeman-Watts operation. One gets a minimal change in personality. If these patients had been less physically incapacitated, I believe they could have gone back to work and been quite effective.

D. Denny-Brown, M.D., *Presiding*
Regular Meeting, Oct. 27, 1955

This meeting, held at the Boston City Hospital, was clinical in nature, and an account of it would be too long for publication.

THE PHILADELPHIA PSYCHIATRIC SOCIETY

Manuel M. Pearson, M.D., *President of the Society, Chairman*
Regular Meeting, Oct. 14, 1955

Practical and Theoretical Applications of the New Drug Therapies in Psychiatry. DR. PAUL H. HOCH, New York.

Chlorpromazine and reserpine are the best known of the current group of tranquilizing drugs. They are so much more effective than any of the others that for all practical purposes they alone need to be discussed, and the following remarks, therefore, are concerned with them exclusively.

If we consider first the group of patients sick enough to require hospitalization, we find that chlorpromazine and reserpine are replacing electric shock therapy in the treatment of acute excited states generally. Apparently, whether the case be manic, schizophrenic, or organic in etiology does not matter, though it is our impression that in cases of senile arteriosclerosis and in postconcussion psychoses chlorpromazine may increase the excitement in some cases and may produce confusion, or increase confusion which already is present. Catatonic cases often do well. In panic reactions and in agitated patients with psychosomatic symptoms sedatives are often useful in combination with chlorpromazine or reserpine.

The medications are useful in very actively deluded and hallucinated patients and may cut down the degree of disturbance, so that in cases with episodic attacks hospitalization may be avoided, though, despite the popular impression to the contrary, chronic disturbed schizophrenics with long hospital records are not being discharged in large numbers. Our impression is that only about 5% of the back ward chronic schizophrenics improve enough with reserpine to leave the hospital, and, while we have no statistics on chlorpromazine, presumably the recovery rate is essentially the same. On the other hand, these drugs have gone a long way toward eliminating mechanical restraint and seclusion and serve as an effective substitute for electroshock in this group.

Psychiatric depressions generally do not respond to chlorpromazine or reserpine, and electroshock therapy is still the method of choice. An exception is the agitated involutional depression,

in which chlorpromazine, especially if combined with metamphetamine, has proved very useful. This statement applies primarily to hospitalized cases in which adequate supervision is available to preclude the danger of suicide.

Little difference apparently exists between the effectiveness of chlorpromazine and reserpine in cases in which either is indicated, but chlorpromazine acts more rapidly than reserpine and is more effective if oral medication is to be used. Chlorpromazine has more side-effects, but in our experience the only serious complication is the development of agranulocytosis. Liver dysfunction and Parkinsonism have responded readily to discontinuation of the medication. Incidentally, we have found that laboratory tests were of no help in predicting the onset of liver involvement. Our observations have not shown any contraindication to the use of the drugs together, and we have some reason to believe that they act synergistically.

The dose which will be required in any given case is unpredictable and shows wide variations. Up to 16 mg. of reserpine and 4000 mg. of chlorpromazine in 24 hours have been used. In trying to determine the effectivenes of the drug in a given case, it is not unreasonable to continue a dosage of 2000 to 3000 mg. of chlorpromazine daily for at least two to three months, assuming, of course, that no complications develop. No evidence has been produced that either chlorpromazine or reserpine is habit-forming. Patients do not demand their continuance, and usually no disturbance follows their complete withdrawal. In rare instances convulsions have occurred when massive doses were terminated abruptly, but a very rapid reduction procedure may be used safely.

The current very widespread use of chlorpromazine and reserpine with psychoneurotic and mild or incipient psychotic ambulatory patients by general practitioners, internists, and others without psychiatric training suggests the need for a word of caution. Failure to establish an accurate diagnosis or to evaluate the case carefully not only tends to produce disappointing results from treatment but, on a more serious level, may lead to the neglect of other treatment which would be specifically indicated, or to the continuance of that which may do definite harm. Like the bromides, of a few years ago, or the barbiturates, more recently, chlorpromazine and reserpine are active drugs which, used inappropriately, may have grave consequences. Particularly is this true in the mild depressions, which may be accentuated by their use to the extent that the patient may become suicidal. Further, depression may be produced as a symptom when reserpine is used for hypertension in patients who showed no such tendency when the treatment was instituted. In some instances the degree of depression so generated has been great enough to require electroshock therapy. Feelings of depersonalization are fairly common with chlorpromazine and may be very disturbing to patients unless they are warned of the possibility in advance. Obviously, such conditioning requires the use of great skill and tact to temper the effects of suggestion.

Conditions which are not grave enough to require hospital care respond to chlorpromazine and reserpine much less dramatically than those which are severer. Yet we know of patients suffering from incipient schizophrenia who have been treated as outpatients with chlorpromazine when without it hospitalization would have seemed the only solution; of patients with mild cerebral arteriosclerosis and senile psychoses, particularly when restlessness and agitation were features of the clinical picture, who were doing better in general than a control group in which barbiturates were used, and of some cases of psychoneurotic phobias responding surprisingly well. In treating the outpatient psychoneurotic cases especially, the feelings of depersonalization mentioned previously must be considered. Occasionally these have been so marked that discontinuance of the medication has become necessary.

The use of chlorpromazine and reserpine has raised very interesting questions about the relationship of these drugs to psychotherapy. Every shred of evidence points to the assumption that both medications afford purely symptomatic relief and that they operate principally in reducing anxiety. Theoretically, since the presence of anxiety serves as a motivation for treatment, the action of the drugs in alleviating it would seem to operate against the psychotherapeutic approach; but in our experience psychotherapy has been facilitated, and we have been impressed by the number of acutely disturbed state hospital patients who, after being relieved of their acute symptoms, have asked spontaneously for psychotherapy. The demand for these services is encouraging, though, unfortunately, it has proved impossible to provide a solution with the present limited personnel. Certainly, we do not see chlorpromazine and reserpine as alternatives to psychotherapy; rather, we regard them as adjuvants which not only relieve distress but act in some way to strengthen the ego functioning of the patient.

Our knowledge of the mode of action of chlorpromazine and reserpine pharmacologically is very fragmentary. Structurally the two have nothing in common; yet their effects in relieving

symptoms are almost identical, and both will counteract such drugs as mescaline and LSD-25 in the psychic field, as well as on the electroencephalogram. By analogy, they bear a marked resemblance to the drugs used to treat epilepsy in that in both instances the symptom is treated effectively, though the causal factors are not altered, and also in that, after a period during which the symptom has been eliminated, often it does not reappear even when the medication is stopped. Theoretically the effect could be due to depression of function at any three levels—the cortex, the midbrain, or the connecting fiber tracts. The fairly frequent occurrence of Parkinsonism suggests that at least part of the action is on the midbrain, and there is some evidence that a clamping down on the activity of the reticular substance may occur, which, in turn, reduces stimulation of the cortex arising from this source. This, in turn, may have some connection with the synthesis of acetylcholine. The question whether these speculations will prove to be justified remains for the future, but in any case additional investigation should prove valuable in gaining an increasing understanding of cerebral physiology. Unlike electric shock therapy, which has proved so completely sterile in gaining insight into mechanisms of behavior, the type of psychopharmacology evolved in the studies of chlorpromazine and reserpine may well furnish the missing link in psychiatry and form a basis for sound therapeutic advances.

DISCUSSION

DR. MORRIS W. BRODY: Chlorpromazine and reserpine might seem to some to be the present-day fulfillment of the wish Freud made 50 years ago for a pill which would eliminate the lengthy procedure of psychoanalysis. Unfortunately, however, our observations lend little support for such an optimistic view. As Dr. Hoch has pointed out, these drugs are much more effective in cases of acute psychosis than in the psychoneuroses, which form the bulk of private psychiatric practice; and, admitting their value in replacing electroshock, in reducing the need for restraint, and in making acutely disturbed patients accessible in hospital cases, the effect on the cases seen in private practice has been to interfere with, rather than to advance, the course of psychotherapy. This is due to (1) the tendency of the patient to regard any tangible agent, such as a medication, as the primary agent and psychotherapy as the secondary agent; (2) the relief from anxiety afforded by the drug, which decreases the patient's motivation for psychotherapy, and (3) the suggestion implied in the prescription for medication that psychotherapy by itself is inadequate. For these reasons, I believe that in a given case the psychiatrist must decide on either medication or psychotherapy but not on a combination of the two; if he feels that medication is essential, he should arrange to have it prescribed by a colleague. Adequate evaluations of therapy still are lacking, and we must remember that these drugs are so new that we do not know how much of their effect is physiological and how much psychological.

DR. LEO MADOW: The investigation of functional psychoses has been advanced greatly by the discovery of substances like mescaline, which will produce symptoms, and others like chlorpromazine and reserpine, which will abolish them. Apparently, the latter act as blocking agents and prevent impulses from traveling from the cortex to the reticular substance which could be translated into clinical terms as a reduction or elimination of the fight-flight reaction. The blocking action would, however, work equally well on impulses going in the opposite direction, a concept which would fit in well with the James-Lange theory that emotions follow, rather than precede, the autonomic response. We must not forget, however, that the drugs we are discussing have no therapeutic effect in the true sense of the term. While they have been effective in dealing with children, particularly those with brain injuries, who are so disturbed by anxiety that they cannot be reached by psychotherapy, in our experience barbiturates have been equally effective. Chlorpromazine and reserpine work better in situations in which the ego control is lost (psychoses) than in those in which it is retained (psychoneuroses). Still to be explained are the depression and depersonalization associated with their use. The improvement in the mescaline-induced abnormal electroencephalograms following their administration raises the question as to whether they act directly on the ganglion cells.

DR. WILLIAM C. WERMUTH: Dr. Brody's position that treatment should consist of either chlorpromazine or reserpine medication or psychotherapy is unwarranted in my experience. I have found the drugs extremely valuable in cases in which hospital facilities are available so that the patient may go into the hospital briefly when the condition requires it. Using this technique, it has been possible to carry a number of patients essentially as office cases where in the past long periods of hospital care would have been inevitable. The advantage of these medicaments over the barbiturates is that the former do not affect the higher centers. Probably this is one reason

that no addiction occurs, though another would seem to be their slower method of action. Since the giving of medication amounts to a gratification of the patient's oral dependency needs, cases in which it is used should be carefully selected. In mild anxiety states it is contraindicated, but in severe anxiety it is often very helpful.

DR. PAUL HOCH, New York: I would agree with Dr. Brody that we do not yet have sufficient evidence by which to evaluate chlorpromazine and reserpine finally and objectively, but I believe there is proof that their effect is due to their action on the central nervous system. Certainly I do not share Dr. Brody's views that therapy should be an "either-or" proposition, or that two therapists should be involved. The same arguments have been advanced in the past with regard to environmental changes and many other treatments. Psychiatrists often have been misled by the assumption that if a patient works through his conflicts with the therapist he will be cured. This, unfortunately, is not true, and we all have seen patients who gained insight and lost ground. Other forms of treatment, including medication, have something to offer. Their effects, however, are almost invariably enhanced by a combination with psychotherapy.

Dr. Madow's comments on the site of action of chlorpromazine and reserpine are most appropriate. These drugs may act by altering local metabolic processes, though this is by no means certain. The evidence favors the assumption that they are blocking agents affecting primarily the motor discharges. Presumably, their action is mostly subcortical and similar to that of a lobotomy, though they probably affect both the cortex and the ganglion cells to a minor degree. Their subcortical activity would, of course, involve the reverberating circuits and thus might function to reestablish the subcortical regulatory mechanism, which may be disorganized in schizophrenia.

To Dr. Wermuth's comments on non-habit-forming qualities of chlorpromazine and reserpine, I should like to add the remark that neither of them has any tendency to produce euphoria.

News and Comment

ANNOUNCEMENTS

Eleventh Annual Schering Award Competition Opens.—Devoted to research and the communication of knowledge in the medical profession, the Schering Award has begun its 11th program for medical students in the United States and Canada.

Students are invited to participate by selecting one of three suggested subjects and submitting papers to the Schering Award Committee, Bloomfield, N. J. Both a $500 first prize and $250 second prize are offered for each of the three subjects. Decisions are made by a group of judges who are authorities in their respective fields. In addition, every participant in the contest receives a professionally useful gift.

The three subjects for 1956, announced by C. J. Szmal, M.D., chairman of the Schering Award Committee, are:

1. The Clinical Use of Adrenocortical Steroids in Collagen Diseases
2. Metabolic Aspects of the Aging Process
3. New Applications of Antihistamines in Medicine and Surgery

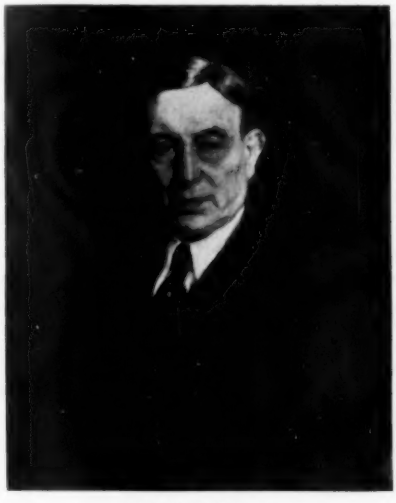
Literature and entry forms are being distributed in the medical schools. Students who are interested in participating, either individually or in teams, should submit their entry forms before July 1, 1956, and manuscripts must be postmarked not later than Sept. 30, 1956.

In a letter to the deans of medical schools, Francis C. Brown, president of Schering Corporation, said the Schering Award Committee was pleased that during the past several years Schering Award-winning papers have been met with increasing interest by professional journals and that many have already been published. The outstanding work of participating students has been such that they will undoubtedly contribute significantly to the professional literature during their careers in medicine, Mr. Brown added.

Obituaries

EGAS MONIZ
1874-1955

Egas Moniz died in Lisbon Dec. 14, 1955, at the age of 81. He was born in 1874 at Avanca, an estate that had been in the possession of his family for at least 500 years. He was graduated in medicine at the University of Coimbra and pursued his studies in neurology in Paris, being appointed to the professorship of neurology at the newly organized Faculty of Medicine at the University of Lisbon in 1911. Here he taught, with intermissions, until his retirement, in 1944. During the intermissions he served as ambassador to Spain, and he signed the Treaty of Versailles for Portugal. He was baptized Antonio Caetano de Abreu Freire, but during his days as a student and pamphleteer took the nom de plume of Egas Moniz, a hero of the Portuguese resistance to the Moors in the Middle Ages. It stuck.



EGAS MONIZ
1874–1955

Being a liberal, he received no support from the autocratic regime that has been in power in Portugal since World War I.

He pursued his studies in the University of Lisbon, and in 1927 published his first work on cerebral angiography. As if this were not enough of a contribution, he undertook studies of human behavior in psychosis that brought him to the conclusion that such repetitive behavior was the expression of abnormal synaptic stabilization in the brain.

In accordance with the theory which we have just developed, one conclusion is derived: to cure these patients we must destroy the more or less fixed arrangements of cellular connections that exist in the brain, and particularly those which are related to the frontal lobes.

Recorded for publication Feb. 7, 1956.

Moniz had the subject well thought out when, at the International Neurological Congress in London in 1935, the subject of the frontal lobe was a main topic of discussion. In addition to anatomic and physiologic studies, the main clinical studies portrayed the serious personality disorders that ensued upon damage to the frontal lobes. Probably no person in the audience other than Moniz foresaw the possibility that controlled damage to the frontal lobes could be of benefit to the human being. Fulton (Frontal Leucotomy and Affective Behavior, Springfield, Ill., Charles C Thomas, Publisher, 1949), after describing the experiments carried out by himself and Jacobsen on the trained chimpanzees Becky and Lucy, describes the episode as follows:

> After the paper was read . . . Dr. Moniz arose and asked if frontal lobe removal prevents the development of experimental neuroses in animals and eliminates frustrational behavior, why would it not be feasible to relieve anxiety states in man by surgical means? At the time we were a little startled by the suggestion, for I thought that Dr. Moniz envisaged a bilateral lobectomy, which, though possible, would be a very formidable undertaking in a human being.* Doctor Moniz had other ideas and within a year he and his associate Almeida Lima, had developed a leucotome, carried out leucotomies on some twenty cases, and published a book on the subject. . . . Although Moniz later stated that the results reported for the chimpanzees convinced him that the operation would be useful in man, we should not forget that he had considered surgical interference long before hearing of our experimental results.

I met Egas Moniz for the first time at this Congress, where we both had exhibits on the use of colloidal thorium dioxide for visualization of the brain. I had previously followed his researches since 1927 in cerebral angiography, but with misgivings, since a safe radiopaque medium was not available at first. His exhibit was of more than ordinary interest, and his procedure was soon adopted to the United States. At that time he made no mention to me of his ideas in regard to leucotomy. However, when his monograph (Tentatives opératoires dans le traitement de certaines psychoses, Paris, Masson & Cie, 1936) appeared, a copy of which was sent to me by Fulton, the import of this discovery was immediate and profound. Within a few months Watts and I reported our first case (Prefrontal Lobotomy in Agitated Depression, *Med. Ann. District of Columbia* **5**:326 [Nov.] 1936).

The genius of Moniz was recognized. An International Congress of Psychosurgery was held in Lisbon in 1948, and the following year Moniz (with Hess) was awarded the Nobel Prize in Medicine.

WALTER FREEMAN, M. D.
Los Altos, Calif.

* Brickner, R. M.: The Intellectual Functions of the Frontal Lobes, New York, The Macmillan Company, 1936. Brickner's patient had had a bifrontal lobectomy by Dandy in 1930 and was described at the same symposium.

Abstracts from Current Literature

EDITED BY DR. BERNARD J. ALPERS

Physiology and Biochemistry

HEADACHE AND HYDRATION. A. M. OSTFELD, D. J. REIS, H. GOODELL and H. G. WOLFF, A. M. A. Arch. Int. Med. **96**:142 (Aug.) 1955.

Two types of edema have been observed in patients subject to vascular headache of the migraine type. The first was a localized fluid accumulation that developed during the headache and occurred principally in those areas of the scalp in which the headache was present. Tenderness and pitting edema were present in this area. The second type was a more generalized fluid accumulation that occurred prior to the headache, involved many parts of the body, and was demonstrable by weight gain and pitting of the dependent extremities.

In this study the authors report the significance of the two varieties of fluid accumulation in migraine patients by visualization of bulbar conjunctival vessels with a slit lamp during 200 migraine headaches. They noted that the presence of local edema never preceded the headache attack, but that as the headache persisted, the edema became more pronounced and frequently outlasted the headache. The edema subsided with the administration of vasoconstrictor agents. At the onset of the headache, the arterioles and vessels were dilated and the number of capillaries was increased on the headache side as compared with the headache-free side. Localized edema and occasional hemorrhages were also present. Vasoconstriction, with clearing of the localized edema, occurred after the intravenous administration of arterenol, and the headache diminished in intensity. The sensitivity of the vessels to the vasoconstricting agent arterenol was increased at the onset of the headache and was greatly reduced during the headache attack. The authors conclude that the local edema at the headache site occurred as a result of vasodilatation, and there apparently is a substance or substances in the fluid accumulation that damaged tissue and lowered deep-pain thresholds. This fluid accumulation is related to the mechanism of migraine headache.

The authors then produced overhydration and widespread fluid retention by the administration of hormones and electrolytes and found that the headaches were not related to weight gain, as they could not prevent or precipitate headaches by reducing or increasing body weight. The authors conclude that nonlocalized edema was neither causally nor mechanically related to the mechanism of headache.

MANDEL, Philadelphia.

THE SOURCE OF FAT IN FAT EMBOLISM. W. W. GLAS, M. M. MUSSELMAN, and H. L. DAVIS, A. M. A. Arch. Surg. **71**:600 (Oct.) 1955.

Three theories have been proposed to explain the mechanism of fat embolism:

1. Fat is mechanically liberated and gains access to open veins. This theory has been widely accepted, but it has been stated that there is insufficient fat in the human femur to produce fatal fat embolism.

2. The humoral theory claims that a stress reaction releases blood lipids from their stabilizing suspension.

3. A third concept assumes that entrance of a small amount of fat into the veins triggers off a breakdown of the physiologic fat emulsion.

Experimental investigations in rabbits gave the following findings:

The fat in the marrow of the femur of one rabbit was sufficient to cause fatal fat embolism when injected intravenously into another rabbit. Fat embolism occurred in a few seconds after fracturing the femur. Its degree was lessened when the rabbits were in hemorrhagic shock. Fat embolism developed in the absence of the adrenal glands; it was not produced by corticotropin or cortisone. Depending on the physical properties of the fat, a significant amount was transported from the marrow cavity of the bone to the lung.

The authors' studies support the mechanical theory of mechanical fat embolism but fail to confirm the humoral and trigger hypothesis.

In the discussion of the paper, L. G. Hermann recommended the treatment of fat embolism with an intravenous solution of 5% dextrose and 5% alcohol in doses of up to 2000 to 3000 cc. daily.

LIST, Grand Rapids, Mich.

NEUROLOGICAL DEFICIENCIES AFTER ABLATION OF THE PRECENTRAL MOTOR AREA IN MACACA MULATTA. A. M. TRAVIS, Brain **78**:155, 1955.

Travis reinvestigated the function of the precentral motor area alone in eight monkeys (Macaca mulatta), since previously reported studies have indicated that the precentral motor area was defined as a functional unit that did not coincide with the previous definitions of the "motor cortex." The motor cortex ablations were performed according to the precentral and supplementary motor maps by means of a suction aspiration technique that was thought to reduce the trauma to the adjacent cortex.

After a precentral ablation, the author noted immediate hypotonia and impairment of voluntary movements in the contralateral limbs, but no ipsilateral defects were found. The hypotonia merged gradually into a state of normal tonus, and a partial recovery of motor performance followed, with movements reappearing as soon at distal as at proximal joints, although impairment was present at both joints. No significant spasticity developed, although there was a transient hyperreflexia, indicating that ablation of the motor cortex alone does not result in spasticity. Within 2 to 12 weeks the animals were able to pick up small stationary objects by apposition of the thumb and index finger, but after maximal recovery this was accomplished with only half-speed as compared with the performance of a normal animal. Placing and hopping reactions returned after one to three weeks in the affected limbs, but they were performed in a regarded hypermetric manner, in animals having unilateral or bilateral precentral lesions. Travis believes that this finding indicates the important role played by the supplementary motor cortex in postural reflexes. The abdominal reflexes on the side contralateral to the lesion were diminished for four to six weeks after operation, but then were active and normal. The plantar reflex consisted of apposition of all toes instead of the normal active flexion of the toes. Atrophy occurred in the contralateral limbs during the period of disuse and completely subsided after maximal recovery was obtained. These results do not support the concept that all parts of the motor cortex exert some control over all parts of the body musculature.

MANDEL, Philadelphia.

NEUROLOGICAL DEFICIENCIES FOLLOWING SUPPLEMENTARY MOTOR AREA LESIONS IN MACACA MULATTA. A. M. TRAVIS, Brain **78**:174, 1955.

The supplementary motor area is defined as a distinct somatotopically organized field by electrical stimulation, and Travis, in this study, determined the effect upon the nervous system of its removal. Her results were obtained by producing lesions in 12 monkeys (Macaca mulatta) which had involvement of the supplementary area alone or from ablations involving either the precentral or the supplementary motor area with injury to the other area.

She reports that unilateral supplementary area lesions produced weak, transient grasp reflexes in the contralateral limbs, without paresis, but with bilateral hypertonia of the shoulders. Simultaneous bilateral ablation of the supplementary area produced little paresis, but posture and tonus were greatly affected. There was an immediate increased resistance to passive movements in the flexor muscles, and within two to four weeks contractures developed. The spasticity demonstrated topographical localization according to the portions of the areas removed. If the supplementary area was removed bilaterally in two stages, at six-month intervals. Travis noted that no hyperactive tendon reflexes appeared, and weak temporary grasp reflexes were found after the second operation. However, when, in addition to bilateral supplementary area ablation, there occurred unilateral injury of the precentral hindlimb area, the immediate resistance and final contractures were less in the lower limb opposite the additional unilateral precentral ablation.

Travis next produced lesions involving unilateral precentral and supplementary limb areas, which resulted in an immediate hypotonic paresis and impairment of tendon reflexes. The period of diminished reflex activity merged into a state of hyperreflexia within two weeks. Increased resistance to passive movements was also encountered in this experiment, which demonstrated that the paralysis observed was greater than after unilateral precentral motor removals alone. In addition, the placing and hopping reactions persistently disappeared in the affected extremities following unilateral precentral plus supplementary motor area lesions. No motor impairment, spasticity, or grasp reflexes were found after removal of the portion of Area 6 not included in the precentral and supplementary motor areas.

The author concludes that the supplementary motor area is a bilateral functioning system that is concerned with control of posture, movement, and the inhibition of release phenomena, and that its removal produces topographical localization of spasticity. Greater deficits are obtained after bilateral ablations than after unilateral ablations. MANDEL, Philadelphia.

VACCINATION-INDUCED POLIOMYELITIS IN IDAHO. L. J. PETERSON, W. W. BENSON and F. O. GRAEBER, J. A. M. A. **159**:241 (Sept. 24) 1955.

The Francis report was accepted in Idaho as sufficient evidence that the Salk poliomyelitis vaccine was safe and potent. The vaccination program began in Idaho on April 19 and was discontinued on April 27. During that period approximately 32,000 first- and second-grade children received one injection of vaccine produced by the Cutter Laboratories.

The poliomyelitis vaccine used in this mass inoculation was found to contain live virus. The first result of the use of such a vaccine was to cause a wave of cases in vaccinated children. The second result was the occurrence of a wave of cases in the contacts of vaccinated children. The widespread isolation of poliomyelitis virus and the uniform distribution of cases indicate that the state has been heavily seeded with virus prior to the normal poliomyelitis season. The effect of this early, unnatural seeding is unpredictable. (The total number of cases as of Aug. 1 was 130. In the six years from 1949 to 1954 there had been an average of 27.2 cases reported by July 1. From Jan. 1 to July 1, 1955, a total of 100 cases were reported.)

ALPERS, Philadelphia.

APPLICATION OF THE FIELD THEORY TO GLIOMATA. L. VAN DER HORST, J. Neuropath. & Exper. Neurol. **14**:369 (Oct.) 1955.

Van der Horst believes that Willis' field theory is applicable to cerebral gliomata. This theory supposes that tumors arise from small or large fields of tissue and enlarge not only by cellular proliferation but also by progressive neoplastic conversion of tissue within these fields. Several cases are presented examination of which disclosed widespread glial proliferations ("gliopathies"). Such gliopathy is believed to be potentially a glioblastoma, and dedifferentiation, when it occurs, causes various degrees of proliferation that can be classified by Broders' original system of grading.

SIEKERT, Rochester, Minn.

THE EFFECT OF ADENOSINETRIPHOSPHATE IN NERVOUS SYSTEM ANOXIA. A. A. KAUFMAN and K. A. BLINN, J. Neuropath. & Exper. Neurol. **14**:387 (Oct.) 1955.

The anaerobic breakdown of adenosinetriphosphate (ATP) to adenosinediphosphate is generally considered to be the universal source of immediate energy for the activities of the nerve, and other, tissue. The resynthesis of ATP derives from oxidative metabolism. ATP was tested for a possible protective action against anoxia of the nervous system.

In mice, parenteral administration of ATP was extremely toxic, producing bradycardia and bradypnea. Although a statistical increase in anoxic survival time was achieved, this "protection" was due, possibly, to cardiorespiratory slowing, with resultant delay of tissue oxygen elimination. However, neostigmine (Prostigmin) did not produce an equivalent increase in survival. A mercurial diuretic prevented the toxic effect of ATP, but anoxic survival was impaired.

Topical application to peripheral nerve of rabbits and to the auditory cortex of cats yielded neither toxic effects nor increased survival of evoked and spontaneous potentials in the presence of anoxia. Intracarotid ATP was very cardiotoxic but ineffectual against anoxia.

SIEKERT, Rochester, Minn.

CONVERGENCE AND INTERACTION OF AFFERENT IMPULSES ON SINGLE UNITS OF RETICULAR FORMATION. M. SCHEIBEL, A. SCHEIBEL, A. MOLLICA and G. MORUZZI, J. Neurophysiol. **18**: 309 (July) 1955.

The patterns of individual units in the reticular formation, mainly of the medulla oblongata, were studied in cats, using microelectrodes. Surface-positive polarization of the cerebellum and vagal, acoustic, and cortical stimulations were carried out.

The authors conclude that the degree of convergence of afferent impulses upon reticular units is widespread but definitely limited, that the patterns of convergence show marked variations among the individual units, that some afferent systems affect extensive connections in the reticular formation, while others appear to make few such connections, and that corticoreticular relations appear to mirror only to a slight extent the organization of the afferent systems to the bulbar reticular formation. They believe it is justifiable to consider that portion of the reticular formation studied as being composed of groups of functionally distinguishable fractions, each of which consists of those cells whose synaptic scale includes terminals from the same afferent modality.

SIEKERT, Rochester, Minn.

CORTICAL RESPONSE TO STIMULATION OF MEDULLARY PYRAMID IN RABBIT. HSIANG-TUNG CHANG, J. Neurophysiol. **18**:332 (July) 1955.

The response of the motor cortex to electrical stimulation of the pyramid in rabbits was found to consist of a rapid, surface-positive deflection and a surface-negative slow wave. These were attributed, respectively, to the activity of the axon and the dendrites of large pyramidal neurons. When the recording electrode was gradually pushed into the depth of the cerebral cortex, the electrical signs of both deflections underwent a series of changes, a reversal occurring in the stratum of large pyramidal cells.

The dendritic component of the antidromic cortical potential could be modified by changing the ambient temperature of the cortex and was completely abolished at temperatures below 22 C. The axonal component was free of the blocking effect of lowered cortical temperatures. Traumatic injury to the cortical surface and anoxia both affected the dendritically generated slow wave more severely than the axonal component.

Subcortical recordings of the antidromic action potential indicated that two main groups of fibers are present in the pyramidal tract, the one conducting at 30 meters/sec. and the other at 15 meters/sec.

SIEKERT, Rochester, Minn.

Psychiatry and Psychopathology

ASSOCIATION OF MATERNAL AND FETAL FACTORS WITH DEVELOPMENT OF MENTAL DEFICIENCY: I. ABNORMALITIES IN THE PRENATAL AND PARANATAL PERIODS. B. PASAMANICK AND A. M. LILIENFELD, J. A. M. A. **159**:155 (Sept. 17) 1955.

It has been repeated frequently in recent years that mental deficiency is not in itself a clinical entity but, rather, is a symptom present in a large number of diseases of varying etiology. Pasamanick and Lilienfeld felt that a proper approach to the question of association of maternal and fetal factors with the development of mental deficiency might be made by selecting a group of retarded children born in a given area and then reviewing the maternal and fetal factors as recorded on birth certificates and hospital obstetrics records. This paper is a report of the results of such an investigation with regard to mental deficiency.

The prenatal and paranatal records of mentally defective children born in Baltimore between 1935 and 1952 (analysis of 639 cases in this report) showed significantly more complications of pregnancy and delivery, prematurity, and abnormal neonatal conditions than a similar number of matched controls. The nonmechanical abnormalities, such as bleeding during pregnancy and toxemia, rather than the mechanical factors of delivery, appear to be important factors in this association.

The authors point out that there is a continuum of reproductive casualty, consisting of brain damage incurred during the prenatal and paranatal periods as a result of abnormalities during these periods, leading to a gradient of injury that extends from fetal and neonatal death through cerebral palsy, epilepsy, behavior disorder, and mental retardation. This continuum has implications for further research in the etiology, diagnosis, management, and prevention of these neuropsychiatric disorders. Any effort toward prevention must of necessity be directed at an improvement of conditions associated with maternal health.

ALPERS, Philadelphia.

Meninges and Blood Vessels

PERIARTERITIS NODOSA. Z. SAGAL, Ann. Int. Med. **42**:711, 1955.

Sagal reports a case of periarteritis nodosa in an 18-year-old mentally defective youth, who clinically had symptoms of mental deficiency, allergic skin reactions, cough and convulsive seizures. A roentgenogram of the chest, taken during a period of observation in the hospital, was suggestive of tuberculosis, although other granulomatous lesions were considered. Enlargement of the liver and spleen subsequently developed, and he had a persistent albuminuria with intermittent urticaria. Cortisone was given, and he developed the signs of excessive hormone administration, consisting of a moon face, edema of the legs, and two generalized convulsive seizures. He gradually developed signs and symptoms of uremia and died. The gross findings at autopsy were an acute necrotizing arteritis of the kidneys, spleen, and liver, as well as a granulomatous lesion of the lung. Diffuse interstitial fibrosis with chronic inflammatory cells was noted in the kidney upon microscopic examination.

Gross examination revealed an edematous brain with relative atrophy of the frontal and temporal lobes. Numerous petechial hemorrhages were present throughout. Microscopic examination revealed changes in the small meningeal and intracerebral arterioles, consisting of acute fibrinoid necrosis of many vessels, as well as chronic subintimal fibrous proliferation of the smaller vessels. Many petechial hemorrhages and small areas of infarction were present. The vessel changes were similar to those found in the liver, spleen, and kidneys, being typical of periarteritis nodosa.

MANDEL, Philadelphia.

COARCTATION OF THE ABDOMINAL AORTA WITH DEATH FROM RUPTURE OF AN ANEURYSM OF A CEREBRAL ARTERY. S. F. KAUFMAN and J. W. MARKHAM, Ann. Int. Med. **43**:418, 1955.

Kaufman and Markham report the occurrence of an aneurysm of the left anterior cerebral artery in a 77-year-old white man having an adult type of coarctation of the aorta. The simultaneous occurrence of these anomalies is a rare condition, particularly when it is associated with a ruptured cerebral aneurysm.

The patient had no previous history of hypertension and suddenly experienced dizziness, which was followed by coma. Neurologic examination revealed nuchal rigidity, without localizing signs, and blood in the spinal fluid. Seven days later he had generalized convulsive seizures, and an arteriogram revealed the presence of a left anterior cerebral aneurysm. He subsequently developed papilledema and died shortly, after unsuccessful ventricular taps. At autopsy a thin-walled saccular aneurysm was found in the left anterior cerebral artery 7 cm. distal to the anterior communicating artery. The aneurysm had ruptured into the adjacent frontal lobe and into the left lateral ventricle, which was filled with clotted blood. This aneurysm was associated with a narrowing of the abdominal aorta, measuring 4 cm. and arising below the origin of the renal vessels.

MANDEL, Philadelphia.

CAROTID EXPLORATION FOR HEMIPLEGIA FOLLOWING MITRAL VALVE SURGERY. D. P. BOYD, J. A. M. A. **159**:112 (Sept. 10) 1955.

Boyd points out that postoperative hemiplegia following operations may be due to obstruction for mitral stenosis. At the time of operation no complications were encountered. No clots were palpable either in the appendages or in the auricle itself. The patient was operated on in mid-afternoon, and in the early postoperative period, during which time special-duty nurses were present, nothing alarming was noted. The next morning the patient did not respond and a hemiplegia on the right was present. It was found that the left carotid artery was not pulsating, and an exploratory operation on the left common carotid artery was performed at once. A fresh blood clot was removed and anticoagulant therapy instituted postoperatively, but the patient died 24 hours after the second operation.

Boyd points out that hemiplegia following operations may be due to obstruction of the common carotid artery or to obstruction of the internal carotid artery at the bifurcation of the common carotid artery. He feels that the anesthetist should continually observe pulsations in the carotid arteries during the performance of the surgical procedure. In every case of mitral valvotomy in which the complication of hemiplegia is demonstrated postoperatively, the carotid artery should be explored. All nurses, residents, and others concerned in the postoperative care of these patients should be thoroughly briefed relative to prompt recognition of this complication.

ALPERS, Philadelphia.

Diseases of the Brain

CUSHING'S SYNDROME. L. W. POWELL JR., S. NEWMAN, and L. W. HOOKER, A. M. A. Am. J. Dis. Child. **90**:417 (Oct.) 1955.

The authors report a case of Cushing's syndrome in a 12-week-old girl. Obesity, mongoloid features, and hypotonia were noted. Bilateral adrenocortical adenomas, massive pontine hemorrhage, cardiac hypertrophy, and hyaline arteriosclerosis of small arteries were found at necropsy. The syndrome is rare in infants and is seldom caused by bilateral adrenocortical adenomas.

SIEKERT, Rochester, Minn.

OTOGENIC BRAIN ABSCESS. M. F. SNITMAN, H. SINGH, J. GLADNEY, and J. R. SOFRANEC, A. M. A. Arch. Otolaryng. **62**:159 (Aug.) 1955.

The authors report two cases of brain abscess which occurred during an acute exacerbation of chronic otitis media. The first case occurred in a 30-year-old white man with a history of otorrhea for two years, who experienced pain over the right postauricular and temporoparietal area, with a temperature of 104 F. A diagnosis of acute mastoiditis was made, and a right radical mastoidectomy was performed. Thick green pus was encountered, which was cultured and reported as yielding a predominant growth of Achromobacter organisms. However, the postoperative course was complicated by somnolence, severe pain over the right side of the head, and papilledema. Reexploration revealed a brain abscess of the right temporal lobe, which was drained, and treatment progressed successfully with oxytetracycline (Terramycin) and chloramphenicol (Chloromycetin). Achromobacter organisms were also isolated from this abscess. The authors emphasize that Achromobacter organisms are Gram-negative nonpathogenic bacilli which are found in treatment-resistant cases of osteomyelitis or chronic urinary tract infections, but were the etiologic organisms in this unusual otogenic abscess.

The second case occurred in a 60-year-old white man who was given penicillin for an acute exacerbation of chronic otorrhea with fever. Following the subsidence of fever with antibiotics, the patient developed acute mastoiditis, which was treated by radical mastoidectomy. Two weeks after this operation, he developed an auditory aphasia with fever, and reexploration revealed a temporal lobe abscess. The patient recovered after surgical drainage and antibiotic therapy. In this case the absence of clinical findings of intracranial pathology was ascribed to the masking effect of antibiotic therapy. This complication must be considered when antibiotics are used in the treatment of chronic otorrhea.

MANDEL, Philadelphia.

CEREBRAL VASCULAR DISORDERS. RUSSELL BRAIN, Ann. Int. Med. **41**:675 (Oct.) 1954.

The author has analyzed a series of 100 hospital patients and 100 private patients, all suffering from cerebral vascular disease, with specific reference to their age distribution and the nature of their lesion. Only 37% of the hospital patients were past 60, whereas 67% of the private patients were above this age. The explanation of this difference is to be found in the fact that patients with acute cerebral vascular lesions tend to be admitted straight to the hospital, and this includes many with subarachnoid hemorrhage due to rupture of an intracranial aneurysm or angioma, who are younger than most patients with chronic effects of degenerative lesions of the cerebral vascular system, more of whom are seen in private consultation. Hypertension was present in approximately 50% of the 200 patients.

The relative frequency of the principal presenting symptoms in the 200 patients is discussed. A special note is made of advances in our ability to recognize the symptoms produced by atheroma or thrombosis of the internal carotid artery.

The author stresses the value of angiography in the diagnosis of cerebral vascular disease. He states that one should not hesitate to use angiography if one suspects the presence of an angioma or an aneurysm, not even in the presence of cerebral vascular disease, if it is necessary to exclude an intracranial tumor.

ALPERS, Philadelphia.

TUBERCULOMAS OF THE CENTRAL NERVOUS SYSTEM. M. SCHWARTZ, R. A. GILMAN, J. S. ROBEY, J. SETTLE, and L. E. PADDOCK, Ann. Int. Med. **42**:1076, 1955.

The authors report four cases of tuberculomas of the central nervous system in young American Indians ranging in age from 19 to 35 years. Three of the four patients had signs and symptoms of increased intracranial pressure with localizing cerebral signs, and the fourth patient had evidence of a spinal cord tumor from clinical studies. Tuberculomas were removed from the cerebral hemispheres in three of the cases, and an epidural tuberculoma was removed from the lower thoracic spine of the fourth patient. In each instance, the tuberculoma had the characteristic microscopic appearance of a granulomatous lesion with Langhans giant cells and an adjacent glial reaction with astrocytes. Acid-fast bacilli were demonstrated in each case with special stains. Three of the four patients recovered, and each was treated postoperatively with streptomycin, aminosalicylic acid, and isoniazid. The authors believe that the daily postoperative treatment should consist of 2 gm. of streptomycin, 16 gm. of aminosalicylic acid, and 10 mg. of isoniazid per kilogram of body weight. The streptomycin was reduced to 1.0 gm. daily after the first six months, and treatment was continued for a minimum of one year. Intrathecal medica-

tion was not recommended. The rationale of this regimen is that further spread of the disease may occur when the walled-off lesion is removed, and such chemotherapy will prevent the development of tuberculous meningitis or other tuberculomas.

MANDEL, Philadelphia.

UNUSUAL CENTRENCEPHALIC SEIZURE PATTERNS, D. A. HOWELL, Brain **78**:199, 1955.

Howell reports unusual seizure patterns in centrencephalic epilepsy which differ from the characteristic myoclonic petit mal, petit mal automatism, petit mal, and grand mal attacks. At times these patterns simulated focal cortical seizures. In each case, there was no evidence of any disorder of the brain, and the electroencephalogram showed the characteristic picture of bilaterally synchronous, three-per-second wave and spike discharges.

The first group was composed of minor seizures in which a variety of motor, sensory, autonomic, or psychical disturbances occurred without any profound loss of consciousness or amnesia for the attack. The patients were able to recollect, and later describe, the events that occurred in the seizure. The minor seizure occasionally preceded a grand mal attack, and it had a special tendency to occur as a first seizure after change in medication. The motor phenomena consisted of sudden loss of power, affecting the arms or legs, or of persistent trembling or jerking movements of all the extremities. Five patients had movements of the head to one side while they were conscious, and another patient attempted to move her head back to the midline after it had involuntarily turned to the side. Sensory seizures were not as common as the motor type and consisted of high-pitched sounds or blurring of vision as an aura prior to the seizure. The autonomic seizures were not rare either as an aura or prior to a major convulsion. Progressive slowing of the heart was noted in one case; pallor and warm prickling sensations of the head were noted in others. A fourth patient had an epigastric sensation of tightness which rose to her throat, and described a feeling of constriction about the chest. Psychical phenomena, such as dream states and hallucinations, were also encountered.

Twelve patients clearly recollected being unable to speak during a minor seizure, and Howell believes that one or more of the nervous mechanisms subserving speech is paralyzed by the discharges in the minor seizures. Two patients with centrencephalic epilepsy had Todd's paralysis, characterized by transitory postictal hemiparesis following the major convulsion.

Many centrencephalic seizures were found which closely mimicked those arising from cortical foci. Howell believes that the EEG may have failed to reveal an additional cortical focus since the low-voltage spike focus may be in some part of the cortex that is not accessible to the electrodes of the electroencephalograph. Another explanation of these unusual attacks may be that the epileptic discharge spreads by contiguity into the surrounding gray matter, to a distant collection of nerve cells. If the discharge arises centrally, the spread by contiguity may be asymmetrical or restricted to an area in the cortex that would lead to an attack mimicking a focal seizure.

MANDEL, Philadelphia.

Diseases of the Spinal Cord

POLIOMYELITIS IN PREGNANCY, FETUS, AND NEW BORN. T. BATES, A. M. A. Am. J. Dis. Child. **90**:189 (Aug.) 1955.

Bates reports the cases of a pregnant woman, near term, who developed poliomyelitis and her offspring, who developed poliomyelitis 40 hours after birth.

The incidence of poliomyelitis appears to be increased in pregnancy, although this increase may be more apparent than real. Fetal poliomyelitis is rare. Poliomyelitis in the newborn is not rare, although the diagnosis may be difficult.

SIEKERT, Rochester, Minn.

PERIODIC PARALYSIS ASSOCIATED WITH HYPERTHYROIDISM. M. A. Linder, Ann. Int. Med. **43**:241, 1955.

Linder reports three cases of periodic paralysis in white men, 25 to 27 years of age, which was associated with hyperthyroidism. In each case, the paralysis was of an intermittent nature, consisting of flaccid paralysis of the muscles of the lower extremities with loss of deep tendon reflexes and loss of response to electrical stimulation. The paralysis persisted from 4 to 62 hours and was reversed by the oral and intravenous administration of potassium salts. In each instance the onset of paralysis of the lower extremities followed the ingestion of large carbohydrate meals, consisting of spaghetti or rice, and could also be precipitated by the intravenous admin-

istration of 200 gm. of dextrose containing 50 units of regular insulin. Each case was also associated with the signs and symptoms of hyperthyroidism, including nervousness, excessive perspiration, and diarrhea with elevated B. M. R. and with elevated radioactive iodine uptake in two cases. Electrocardiographic changes, consisting of lowered T-waves, S-T-segment depression, and Q-T interval prolongation were compatible with hypopotassemia. Thyroidectomy in each of these cases resulted in spontaneous absence of paralysis and inability to provoke attacks.

The author believes that the attacks of periodic paralysis in hyperthyroidism are related to carbohydrate metabolism, particularly hyperglycemia, since this part of the metabolic process is most profoundly affected by the overactive thyroid gland. There may be an abnormal requirement of potassium during the metabolic process of glycogenesis, leading to low serum potassium levels and paralysis. However, with the cure of hyperthyroidism, there is a disappearance of the paralysis.

MANDEL, Philadelphia.

THE CLINICAL EPIDEMIOLOGY OF POLIOMYELITIS. D. HORSTMANN, Ann. Int. Med. **43**:526, 1955.

Horstmann emphasizes the family epidemiology of poliomyelitis, for the high rate of infection among household members suggests that contact within a family is conductive to the spread of infection. In addition, the family as a unit is infected rather than the usual pattern in other diseases, such as measles, in which secondary cases follow the primary case after a characteristic time interval. By use of the complement fixation test, which reveals a highly significant titer of infection, the author was able to define more clearly the spread of poliomyelitis throughout families. She reported two cases in which the epidemic centered about a nursery school and the family contacts of the nursery school children. In one of these cases, the adults, as well as the children, were susceptible to the disease, with the father developing Type 1 paralytic poliomyelitis.

The pattern of these two "nursery-school-borne" epidemics emphasizes one of the striking features of the epidemiology of poliomyelitis, for in the past 20 years in the United States there has been a shift in the age distribution of the disease, with a steady decrease in the percentage of cases occurring in young children and a continually increasing percentage in adults. Factors such as the strain of virus responsible for the disease, amount of virus introduced into the body, and the immunity of the host may explain paralysis and death in some cases or mild inapparent infections in others. Adults, however, appear to be more susceptible than young children and develop serious infections, with a high mortality. This pattern has also been observed in adults who develop other virus diseases, such as measles and hepatitis.

MANDEL, Philadelphia.

PROGRESSIVE MUSCULAR DYSTROPHY WITH EXTENSIVE DEMYELINATION OF THE BRAIN. G. A. JERVIS, J. Neuropath. & Exper. Neurol. **14**:376 (Oct.) 1955.

Jervis reports on five members from a closely inbred kindred showing the typical muscular manifestations of the "childhood type," or "Duchenne type," of progressive muscular dystrophy. At least two of them manifested, in addition, mental deterioration, epilepsy, and progressive spasticity, and pathologic study of one so affected disclosed an extensive chronic demyelinating process of the centrum semiovale, similar in character and distribution to that observed in some forms of diffuse sclerosis of the brain.

SIEKERT, Rochester, Minn.

PALSY OF CONJUGATE EYE MOVEMENTS IN POLIOMYELITIS. E. ADLER, J. LANDAU, and E. RABINOWITZ, Confinia neurol. **15**:154, 1955.

Adler, Landau, and Rabinowitz report four cases of paralysis of conjugate gaze occurring in children under 5 years of age. Case 1 was a child of 3 years who had a febrile illness with no neurologic disorder except for paresis of conjugate gaze to the right. Eight days later a remarkable degree of improvement had occurred, and four weeks after onset all eye movements were normal. Case 2 was a child of 4 years who on the sixth day of a febrile illness developed paresis of conjugate gaze to the left and weakness of the left gluteus maximus. The eye signs cleared in two weeks. Case 3 was a child of 3 years who became febrile and somnolent and had spinal fluid findings consistent with poliomyelitis. The virus was recovered from his stool. He failed to follow fingers to either side but had limited vertical movement of the eyes. When the fever subsided, after five days, the eyes were fixed with the gaze straight forward, the vertical movements were not disturbed, and the closing of one eye made some lateral movements

of the open eye visible. The child tilted his head to the left in a torticollis-like position when asked to look to the right side. During the following weeks a progressive improvement began, starting with recovery of left lateral gaze. Full recovery of ocular movements did not occur until four months after onset of the illness. Case 4 was a child of 14 months who developed fever and drowsiness. When the temperature dropped, he was found to have paralysis of lateral gaze to the left. "In closing one eye distinct lateral movements of the open eye could be elicited." The patient recovered within a few weeks, with only a slight residual weakness of the left lateral rectus muscle.

The authors review the rather scanty literature on the subject and conclude that the disturbance is more frequent in poliomyelitis than is generally assumed.

FOLEY, Boston.

Peripheral and Cranial Nerves

BELL'S PALSY. D. TAVERNER, Brain **78**:209, 1955.

Taverner reports his findings in the study of 100 cases of Bell's palsy which were evaluated both clinically and electromyographically. The electromyogram was used as a basis in dividing these cases into "successes" and "failures," for the electromyogram consistently demonstrated fibrillation activity, which was indicative of the presence of denervation in the "failure" group. There were 45 recoveries and 55 failures in this series, no sequelae being found in the former cases, but associated movements, contractures of the face, and crocodile "tears" were the sequelae observed in the failure cases. In the successful group, when recovery began, it became evident within 3 to 21 days after the onset of the paralysis and was usually complete in 18 to 150 days. In the failure group, none of the patients recovered within six months to three and one-half years. The age, sex, and clinical features of the failure group did not differ significantly from those in the completely recovered group.

Electromyographic analysis revealed the presence in all 55 cases of blink bursts, which consisted of grouped motor unit potentials synchronous with blinking of the eyes. Associated movements, such as involuntary contraction or overaction of one part of the face during voluntary contraction of another part, and blink bursts were shown to be due to branching and misdirection of the regenerating axons. Taverner states that these findings indicate that in many patients the lesion causing the denervation is of such severity that it cannot be attributed to compression of the nerve in the Fallopian canal. The etiology of Bell's palsy remains obscure, but the pathological basis for this disease is probably a severe, widespread parenchymatous neuritis. The prognosis of Bell's palsy can be accurately determined by electromyographic findings.

MANDEL, Philadelphia.

Treatment, Neurosurgery

ADRENAL SURGERY FOR PSYCHOSES ASSOCIATED WITH CUSHING'S SYNDROME. FRANK C. HAMM, A. M. A. Arch. Surg. **71**:617 (Oct.) 1955.

Two cases of the Cushing syndrome associated with psychosis are reported. Both patients were women exhibiting mental disturbances severe enough to require institutional care. Endocrinologic studies revealed characteristic clinical signs of hypercorticalism. In both instances an adrenal tumor was demonstrated by roentgenologic methods and diagnosis of cortical adenoma verified by operation. Following removal of the lesion, the psychotic symptoms cleared up completely and the endocrine signs showed striking improvement.

LIST, Grand Rapids, Mich.

THE CONTROVERSIAL USE OF CERVICAL SYMPATHETIC BLOCK IN APOPLEXY. G. DE TAKATS, Ann. Int. Med. **41**:1196 (Dec.) 1954.

The rationale for the basis of cervical sympathetic block in apoplexy is that it decreases cerebrovascular resistance, a factor which must be of benefit to the infarcted brain. The author disagrees with the previous observations of Kety and Schmidt, who found that stellate ganglion block causes no change in the cerebrovascular resistance. De Takats states that the former technique shows no change in the femoral artery blood pressure, while stellate ganglion block relaxes the internal carotid artery wall, causing a drop in blood pressure.

In this series, 55 patients with apoplexy were treated by blocking injections of the cervical sympathetic trunk. They received from 1 to 10 injections daily, and five were given bilateral

blocks. A total of 109 injections was given. Injection was given only once if there was a complete regression of neurological symptoms. There were failures in 55% of the group of patients with cerebral infarcts and no failures among the 14 patients having cerebral emboli.

The author believes that the large number of failures does not represent worthlessness of this procedure, since many patients obtained dramatic improvement. The cervical sympathetic block is of help only if the infarct is surrounded by a zone of edema and vasoparalysis, if the cerebral vessels are not unduly sclerotic, and if the circle of Willis is able to act as an effective shunt when one internal carotid artery or one of its main branches is blocked. In addition, if hypotension is present, revascularization of the ischemic infarct by sympathetic paralysis cannot take place. De Takats states that because the technique is harmless and the results gratifying, further study and use of this method are indicated.

MANDEL, Philadelphia.

CORTISONE IN IMMEDIATE THERAPY OF APOPLECTIC STROKE. H. I. RUSSEK, A. S. RUSSEK, and B. L. ZOHMAN, J. A. M. A. **159**:102 (Sept. 10) 1955.

Various attempts have been made to improve the cerebral circulation in patients with apoplectic stroke, in the hope of decreasing neurological residua. The uncertain responses with such methods have left no alternative to expectant therapy. Inasmuch as cortisone has been found of value in the rehabilitation of hemiplegic patients, through its action in enhancing motivation and promoting a sense of well-being, the authors considered it logical to make a trial of the drug for the acute cerebrovascular accident. Moreover, since local cerebral edema is believed to be a contributory factor in the perpetuation of neurological signs, and since cortisone has been shown to retard the mesenchymal tissue response to various forms of trauma, additional benefit from cortisone seemed possible in this respect.

Thirty-five cases of apoplectic stroke due to cerebral thrombosis or embolism were treated with cortisone within 48 hours after the onset of symptoms. Hypertension was present in 24 of the 35 patients. Diabetes mellitus of mild to moderate severity was present in six patients. In most instances 300 mg. of cortisone was administered orally in divided doses on each of the first two days, with progressive diminution to a maintenance dose of 50 mg. daily through the third week. In addition, when possible, each patient was given a low-salt diet, with 3 gm. of potassium chloride orally each day in divided doses.

Cortisone produced striking effects in 21 of the 35 patients under this regimen, and remarkable neurological recovery had occurred in 27 patients by the end of the third week of cortisone administration. No significant untoward effects from cortisone were noted in this study. Russek and his co-workers consider cortisone a valuable addition to the therapy of acute cerebral thrombosis or embolism.

ALPERS, Philadelphia.

Encephalography, Ventriculography and Roentgenography

ROENTGENOLOGIC ASPECTS OF CHORDOMA. J. R. UTNE and D. G. PUGH, Am. J. Roentgenol. **74**:593 (Oct.) 1955.

Utne and Pugh analyzed 72 cases of histologically proved chordoma which they found in the files of the Mayo Clinic. In 41 of the 72 cases original roentgenograms were available for viewing. Chordoma is a slow-growing malignant tumor arising from vestiges of the embryonic notochord. In only 1 of the 72 cases was a metastatic lesion found and verified. In this one case a metastasis to the scapula was found originating in a sacrococcygeal chordoma. In two other patients the clinical course suggested the possibility of metastasis, but no confirmation was obtained.

Of the 72 chordomas, 18 were located intracranially. Ten were located in the cervical region of the spine. Four were found in the thoracic and lumbar regions, and forty were located in the sacrococcygeal region of the spine. The intracranial chordomas originated near the midline at the base of the brain. Usually the first symptoms were those of increased intracranial pressure; later the symptoms of pressure on adjacent structures were produced. The roentgenographic findings of bone erosion at the base of the skull, in the region of the clivus, and direct or indirect evidence of a soft tissue mass in the same location were almost always present in the cases of cranial chordoma. It is difficult to differentiate chordoma from other intracranial neoplasms because there are no specific symptoms. Chordoma must always be considered in the differential diagnosis of a midline tumor at the base of the brain.

The presenting complaints and roentgenographic findings of chordomas occurring in the cervical, thoracic, and lumbar regions of the spine did not help to differentiate them from other tumors of the spinal column. In most cases a soft tissue mass could be visualized, and usually some destruction of the vertebral bone was also a prominent feature. The sacrococcygeal chordomas produce symptoms which are caused by pressure on the adjacent extravertebral structures or on the emerging nerves. Symptoms referable to the rectum and bladder are often present. Pain is the most frequent early complaint. The pain usually is in the region of the coccyx or rectum and in the average case has been present for three years before admission. Fourteen of the authors' patients had been treated surgically for hemorrhoids in an effort to relieve the rectal pain. Bone involvement was demonstrated roentgenographically in 75% of the cases of sacrococcygeal chordoma for which films were available, and a soft tissue mass was demonstrated roentgenographically in 85% of them. The area of bone involvement typically began in the midline and involved the fourth and fifth sacral segments. As the tumor became larger, more of the sacrum was involved with bone destruction. Calcification was sometimes noted in the soft tissue mass. The finding of a soft tissue mass and bone destruction in this region of the spine, when taken with the prolonged clinical course, allowed the correct diagnosis to be made preoperatively in 40% of the cases studied.

Utne and Pugh found that roentgenographic examination in all the cases of chordoma for which films were available showed that evidence of bone involvement or a soft tissue mass was present in more than 90% of them.

WEILAND, Grove City, Pa.

ROENTGENOGRAPHIC STUDY OF THE VARIATIONS IN THE NORMAL ANTERIOR CEREBRAL ARTERY. A. A. MORRIS and C. M. PECK, Am. J. Roentgenol. **74**:818 (Nov.) 1955.

Morris and Peck selected for presentation a group of 100 patients for whom cerebral angiograms had been performed in the course of investigation of their neurological illness. After complete investigation it was decided that 71 of the 100 patients had neurophysiologic disease without structural change in the brain. It was thought that these patients could be used to study the normal course and appearance of the anterior cerebral artery, and the normal was defined as the pattern that exists in the majority of cases studied. Only patients between the ages of 15 and 45 years were used in this study.

The authors studied the anterior cerebral artery in the lateral view, using the "head tilt" position. The course of the anterior cerebral artery presents a high percentage of variations in normal subjects. Arteriography combined with pneumoencephalography makes for less confusion concerning any possible significance of the normal variations of the course of the anterior cerebral artery in different persons. The configuration of the lateral ventricles and their distance to the artery are often reassuring where small variations in the course of the artery when taken alone might produce some doubt as to whether an abnormality might be present.

Morris and Peck have arbitrarily divided the course of the anterior cerebral artery into four parts. The first part begins at the derivation of the vessel from the internal carotid artery and ends before the vessel encloses the genu of the corpus callosum. In 82% of the cases studied the first part ran anteriorly, almost parallel to the superior orbital plate, but usually presented a convexity directed superiorly and posteriorly. The second part of the artery surrounds the genu of the corpus callosum. In 60% of the cases this part described a narrow curve, with the convexity directed anteriorly, although a wide curve was found in 17% and an acute angulation in 22% of cases. The third part of the artery is defined as beginning after the curvature of the second part is completed, continuing until the first sign of tortuosity in the pericallosal artery is noted. In 84% of the cases the third portion of the artery formed a round and smooth course with its convexity upward. The fourth portion of the artery begins at the end of the third portion and extends to the most distal part of the vessel. Morris and Peck further subdivide the fourth part into three major categories, in each of which they describe a number of variations. The variations are so numerous that the authors conclude that the course of the fourth part is not reliable in diagnostic significance.

Bilateral filling of the anterior cerebral arteries has been obtained from unilateral injection in 38% of cases. In these cases it was noted that when a variation in the course of the anterior cerebral artery occurred it was almost always a symmetrical bilateral occurrence. For this reason the authors now routinely perform bilateral cerebral arteriography to study the course of both anterior cerebral arteries. When this procedure is combined with pneumoencephalography, a high degree of accuracy can be expected for lesions near the normal course of the anterior cerebral artery.

WEILAND, Grove City, Pa.

Books

Hypertension: Humoral and Neurogenic Factors. Ciba Foundation Symposium. Edited by G. E. W. Wolstenholme, O.B.E., M.A., B.Ch., and Margaret P. Cameron; assisted by Joan Etherington. Price, $6.75. Pp. 294. Little, Brown & Company, 34 Beacon St., Boston, 1954.

This symposium on hypertension of the Ciba Foundation is another one of the symposia under the able directorship of Dr. Wolstenholme and the Board of Trustees of the Ciba Foundation. The aim of intimacy and informality throughout the conference is easily discernible throughout the volume. The subject of hypertension, being both pertinent and full of such vagaries, is ably handled in this symposium. The contributors number thirty-three, coming, as they do, from all parts of the world. As is so often true in these informal symposia, the verve of the proceedings comes usually not from the paper itself but, rather, from the warm discussion that follows. Here one sees the workings of the mind of the individual contributor and his skill with the verbal rapier around the conference table. "Dr. I. H. Page of the Cleveland Clinic presents data that the examples of altered responsiveness in neural control of hypertension show that neural regulation of circulatory function is exercised in large part by mechanisms which control and inhibit and do not exclusively work, as previously thought, by means of excitation." Dr. Page goes on to say that the substance is increased in the blood of some renal hypertensive animals and of some patients with advanced hypertensive disease. Page and his group go on to say "cerebrotonin," pertaining to some mythical substance from the brain liberated into the blood, to which is ascribed pressor activity, pending identification, is called by this name. Unfortunately, he himself admits that the name given to this pseudosubstance is not that of a substance, but, rather, of a phenomenon and that "to give it a name is both fashionable and convenient," but a perfect mirror-image of what we already have too much in medicine today. The vasoconstrictor serotonin has been finally identified as 5-hydroxytryptamine. It is apparently a widely distributed natural pressor agent and is identical to that isolated by G. Erspamer, of the University of Bari. The latter gave his vasoactive material the name "enteramine." Prof. C. Heymans, in pursuing his work on the sinoaortic wall, presents excellent findings emphasizing the fundamental role of the sinoaortic wall in the reflex regulation in homeostasis of blood pressure and suggests that decrease of tone and resistance to stretch of the sinoaortic arterial wall could be the primary mechanism of hypertension. Professor Heymans further showed that drugs such as epinephrine and arterenol in vasopressin contract the arterial walls in the carotid sinus, increase their intrinsic tension, and decrease their distensibility, causing stimulation of the carotid sinus nerve receptors. This stimulation then induces a reflex fall in systemic blood pressure and decreases or suppresses the hypertensive reflexes normally provoked by decreasing the blood pressure in the carotid sinus. Drugs that relax the arterial walls, such as the various adrenolytic drugs—epinephrine and dibenamine—increase the distensibility of the carotid sinus and decrease the stimulation of the pressorreceptors and thus induce a reflex rise of systemic arterial pressure. If this be true, and Professor Heymans' data are very convincing, it is certainly possible that biological conditions in the arterial vessels in young persons may well occur without any pathological changes being seen at necropsy. If so, it might account for some cases of essential hypertension, although Professor Heymans does not make any such claim. Dr. George Perera, of Columbia University, discusses hypertensive disease without hypertension, and his paper serves again to focus attention on a very important aspect of hypertension, as suggested by the title. He describes three periods in the life of hypertensive patients in which there may well be an associated normal blood pressure. The first is prior to the development of hypertension; the second is one occurring after the appearance of the hypertensive disease, and, the third, in which hypertension may have vanished, is presented in a series of six unusual cases in which papilledema and retinopathy appeared during a period of sustained normal blood pressure when they had been absent prior to the episode. Although Dr. Perera admits that the clinical observations he presents require further documentation, it is well known that the chairman of this symposium, Dr. G. W. Pickering, at St. Mary's Hospital, London, has suggested on many occasions that a mechanism of malignant hypertension is closely related to blood pressure levels, particularly to increases above the critical level for each patient. If so, the presentation of these six cases indicates that in these cases other mechanisms must certainly be

operative. But if the complications of hypertensive vascular disease do not occur with elevations of the arterial blood pressure, then to what (and this is the fundamental question) are the complications, if such they may be called, due? Another fundamental question arises: Is the mere reduction of blood pressure a sufficient therapeutic objective in and of itself? Dr. Grollman has hit the nail on the head when he advocates that the use of the words hypertensive cardiovascular disease be limited to a specific and definable clinical and experimental syndrome and not be used to designate every observed elevation in blood pressure. Drs. Jimenez-Diaz, Barreda, and Molina report that arternol (nor-adrenalin) is formed in the arterial wall itself and further suggest that the artery is not a simple container, but an active organ. J. H. Gaddum, of Edinburgh, has gone through a hunt for drugs antagonistic to 5-hydroxytryptamine and discusses these very briefly, and Erspamer has made an exhaustive quantitative estimate of the same agent in various tissues of vertebrates. Erspamer feels very strongly that serum 5-hydroxytryptamine originates from the enterochromaffin cells and that it shows no relation whatever to that of the arterial digestive tubes in his experiments. Certainly, from his complete calculations on various animals, the product is very widely distributed among the vertebrates and the ascidians. Dr. Erspamer further supports the hypothesis that this agent is a hormone designed to control renal function via intrarenal hemodynamics. In the discussion the biosynthesis of 5-HT, which Page called serotonin, apparently is a very rapid process, its source being l-trytophan, which, by a process of oxidation and then of decarboxylation by respective enzymes, is formed into the definitive agent. Goldenberg, of Columbia, discusses some chemical screening methods permitting an easy differentiation of pheochromcytoma with persistent hypertension from essential hypertensive vascular disease. Peart, of St. Mary's Hospital, London, discusses persistence of hypertension after removal of a pheochromocytoma and discusses such cases in relation to animal experimental observations, correlating them with human hypertension and showing that there exist extrarenal factors of great importance in the etiology of hypertension. Dr. Grollman, of Southwestern Medical School, Dallas, Texas, presents an experimental basis for what he calls "a renal deficiency theory for the etiology of hypertensive vascular disease and postulates the defect as one of the incretory functions of the kidney." Dr. Govaerts, of Brussels, in discussing vasoconstrictors in renal hypertension, cannot support the hypothesis that during the stage of current hypertension the ischemic kidney releases into the blood any substance with a direct and immediate constrictor action other than on peripheral blood vessels or that in the course of experimental hypertension there is a substance released by the kidney that potentiates the constrictor action of circulating epinephrine. Dr. Helmar, of Indianapolis General Hospital, presents evidence for a sustained pressor principle, which may be the form in which renin is released by the kidney into the blood stream. M. A. Floyer, of the London Hospital, discusses how permanent changes occur in the intact kidney in experimental animals, rendering that same kidney incapable of restoring the blood pressure to normal after removal of the opposite, clipped kidney, and further postulates that the development of vascular lesions caused by the hypertension may contribute to this gradual change in the function of the intact kidney. Dr. Bing, of Copenhagen, discusses how cortisectomy will decrease blood pressure for short periods of time in experimental hypertension, but only in the early stages of the disease. Dr. McMichael suggests that mechanical factors in the production of papilledema and arteriolonecrosis have probably been overstressed in hypertension and that some other agency is damaging the blood vessels; although high blood pressure may contribute to the damage, it is not its sole cause. Dr. J. Genest discusses the relationship of sodium, arterial hypertension, and the adrenal glands and makes a point that a study of seven patients with restricted sodium intake showed the fall of blood pressure to normal in five of the seven patients, but there was no significant modification of any of the urinary steroid fractions studied. Dr. Etienne-Martin, of the University of Dijon, discusses adrenal insufficiency in hypertensives who had bilateral sympathectomy and unilateral adrenalectomy. Professor Braun-Mendendez, of Buenos Aires, discusses fluid and electrolytes in experimental hypertension, and a similar subject is discussed by J. M. Ledingham, of the London Hospital. It is suggested that there is a common underlying disturbance of sodium distribution in heart muscle, and whether this disturbance occurs in the smooth muscle of the arterial wall, and thus produces vasoconstriction, is as yet unknown. Dr. Martini, of the University of Bonn, discusses the importance of the sodium and chloride ions together, and not in their separate states, as the active agents for the production of hypertension and postulates that possibly the membrane of the cell itself is a critical site of activity of the sodium chloride molecule.

As is usual in these symposia, the Herculean task of circumspection belongs to the chairman, Dr. Pickering in this case, and the usual large number of bravos should be accorded him in correlating this tremendous variety of seeds that the four winds have taken into the four corners with ideas that on the surface seem so diverse that it would seem that the common etiology of hypertension could never be postulated. Chairman Pickering emphasizes that the definition of hypertension must remain based on the elevation of blood pressure, it being recognized that the definition is an arbitrary one and that it varies from day to day in the individual, even in the course of his own disease. Pickering goes on to say that more than one mechanism, of course, can produce hypertension and that if it does produce hypertension in a particular set of circumstances, it does not follow that an identical mechanism holds in an entirely different set of circumstances. Since essential hypertension is a negative diagnosis, it is particularly germane to bring this entity into the discussion. He further raises the question whether the impulses arising in the carotid sinus and the aortic arch are a set of reflexes that vary in hypertensive patients and are contributory to the receptor mechanisms in being primed for hypertension. The question of neurogenic hypertension has come in for considerable pummeling in this discussion, and the serious question that remains to every practicing physician is how much of a component does exist in the particular patient he is examining at a particular time and what measures can he use, including drugs, to detect the size of this neurogenic component. Many of these components and modifying factors are discussed in the symposium and in Chairman Pickering's closing remarks. Any comfortable feeling regarding neural factors in the production of hypertension is easily dissipated by the time one closes the covers of this symposium. The variety of influences on the innumerable factors and the number of factors themselves seem to be rising with the number of investigators put forth to prey upon the disease. Chairman Pickering further places 5-hydroxytryptamine or serotonin in its proper perspective by saying that it does not seem to have much to do with hypertension, but probably does have something to do with the control of renal function, and also with keeping practitioners sane. Renin is still under a cloud and does not come out from under it in this symposium. The question of the cortical steroids will probably be better elucidated in future years, when the assay methods are improved and identification is more easily carried out in the various laboratories in regard to the respective function of these substances in hypertension.

The trustees of the Ciba Foundation have once again produced a volume that will provoke much discussion for a long time to come, and, as is usual with these volumes, tremendous value is obtained by the reader who spends time in general discussions following the various papers. Certainly all those interested in any phase of hypertension whatsoever will derive a definite benefit from the provocative ideas in this little volume.

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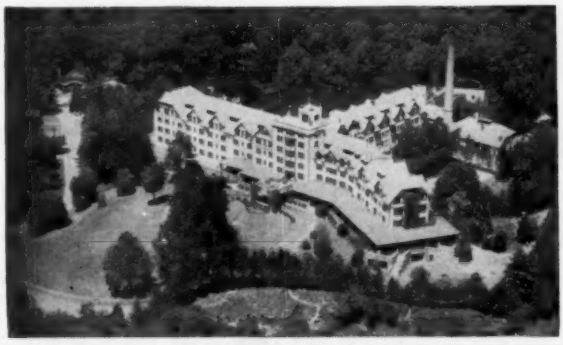

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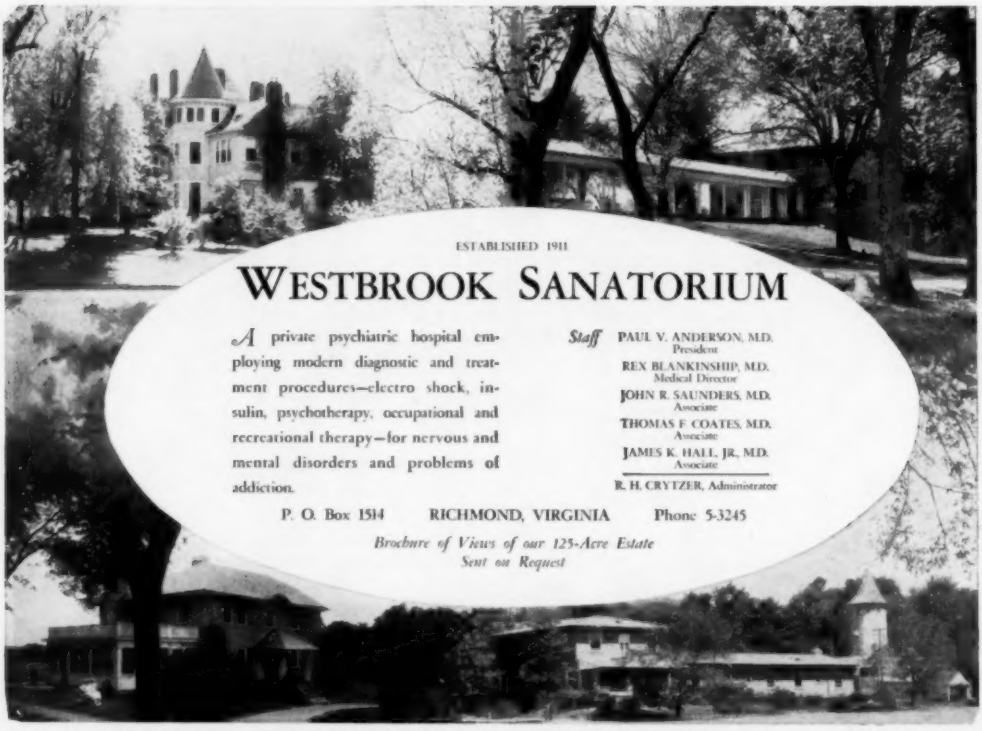

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